THE HEART AND ITS DISEASES:-

WITH THEIR TREATMENT.

BY

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TO

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LINACRE PROFESSOR OF ANATOMY AND PHYSIOLOGY IN THE UNIVERSITY OF OXFORD,

IN

GRATEFUL REMEMBRANCE OF REPEATED ACTS OF KINDNESS AND ENCOURAGEMENT,

THIS WORK IS DEDICATED,

BY

THE AUTHOR.

PREFACE.

WHILE our literature on Heart Disease contains the systematic works of Hope, Stokes, Walshe, and our American *confrère*, Flint, it may seem to indicate some lack of diffidence to venture another work on the subject. While, however, we have such work as Claude Bernard, Cyon, Thirry, von Bezold, Ludwig and Rutherford are doing in clearing up the important subject of cardiac innervation; Pettigrew investigating the heart's structure and evolution; Brunton rigorously testing the action of remedies upon the heart; Richardson paving the way for a better comprehension of the clinical significance of the heart's objective symptoms; Quain searching into its pathological changes; while Peacock is accumulating a store of information on its malformations; and, finally, Geo. Johnson demonstrating the changes in the arterioles in Bright's disease, and their effect upon the heart, and thus giving force to the views of Ludwig and Traube; it is possible that scientific progress may have made room for a newer treatise. In such belief the writer lays this work before the medical public; and, if in it many references are made to foreign authorities to the apparent disparagement or neglect of the workers in his own country, it is in no such spirit, however; nor is the work done in England of inferior importance to the pathological researches of the Germans, and especially their minutely exact following out of the consequences of obstructed circulation. The importance of alterations in arterial tension, and the serious results of venous congestion have been clearly shown by Teuton workers. The gravity of tricuspid imperfection, as

pointed out by Peyton Blakiston in our own country, has especial force given to it by recent research; and the effect of heart failure on the kidneys, and the production of interstitial nephritis therefrom, is now more fairly comprehended. The importance of Bright's disease in the production of heart disease is now being fully recognized, and the crude opinions of Bright and James are being elaborated by Traube, Ludwig, and Geo. Johnson. The introduction of a systematic chapter on the relation of heart disease and kidney disease to each other is novel, and, though the writer's attention has been directed to the subject for years past, the present chapter is rather to be regarded as tentative than conclusive—as inviting more attention to the subject than suggesting the exhaustion of it. Finally, the writer must acknowledge the aid derived from the recent treatise on Heart Disease (Lehrbuch von Herzkrankheiten), of Dr. T. von Dusch, Professor of Medicine in the University of Heidelburgh, who also has kindly permitted the use of his plates; which, along with others from Rindfleisch, will do much to illustrate the subject under discussion.

London, *August 6th*, 1872.

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ERRATA.

Page 7, 6 lines from top, *for* "driving," *read* "diving."

„ 14, 16 lines from bottom, *for* "ectaire," *read* "cataire."

„ 45, 9 „ „ *read* "branches of the venæ cavæ."

„ 71, 19 „ „ *read* "clinically" for "chemically."

„ 79, 5 lines from top, under Percussion, *read* "outwards" *for* "towards."

„ 285, *for* ANÆMIA *read* URÆMIA.

THE HEART AND ITS DISEASES.

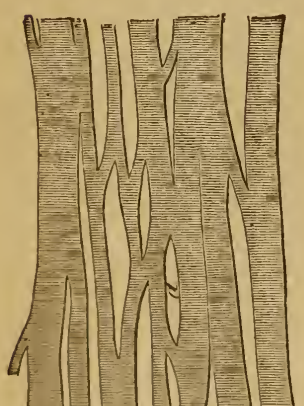
CHAPTER I.

THE HEART—ITS EVOLUTION—WORKING—NUTRITION AND BLOOD-SUPPLY—ITS SLEEP—NERVE SUPPLY AND MODE OF ACTION.

THE heart is a hollow sphere of involuntary muscular fibre, and in its primitive form is a mere pulsatile sac. In its gradual evolution up the scale of creation it becomes *pari passu* more and more complicated, consists of several chambers, and has its force economised and directed by folds or valves, which prevent regurgitation. This provision of valves is not confined to the heart, but is found also in the systemic veins, and in the intestinal canal. In the heart the valves are formed regionally near each other, for the heart itself is merely a mass of involuntary muscular fibre-tubing folded on itself. Pettigrew has shown that the heart consists of seven layers of muscular fibres; the central fibres of the muscular wall are circular, while the fibres towards both outer and inner surfaces pass rather from base to apex, twisting spirally in their course. The outer and inner fibres are continuous into each other, thus the innermost layer 1 is continued into the outermost layer, 7, the next innermost, 2, with the next outermost, 6, and so on. It is, as described by B. W. Richardson, "a coiled spring" of muscular fibre, under a beautiful nerve supply, as we shall see. These fibres are not confined to one-half of the heart, but are mostly common to each, like the figure of 8, only not quite so simple. These fibres have been unwound by Shearle, Pettigrew, and others, who show that some of them are very complex, and

pass into all the four divisions of the heart. This is what might be expected from the gradual evolution of the heart; as Pettigrew has shown that the right ventricle is merely a doubling over of a portion of the primitive left ventricle. In consequence of this community of certain fibres the heart's contraction is almost synchronous, and has lost, to a large extent, the vermicular action, which belongs to involuntary muscular fibre-tubes. But still it is really vermicular, and the synchronicity of contraction in the various fibres is merely a rapid contraction passing from one layer of fibres to the other, as first shown by Schiff, and confirmed by Valentin. The contraction commences first in the auricle, and passes swiftly into the ventricle. The auricular contraction is first excited by a sensation of fulness, and its contraction in its turn excites ventricular contraction by the additional blood thrown into the ventricle by the auricular contraction; the fibres of the auricle contracting in the direction of the ventricle. This is the mode of action of the working. For though a heart will continue to contract rythmically when cut out of the body, especially in the so-called cold-blooded animals, still an effect is produced by the sensation of distension, and both Ludwig and von Bezold have found that increased pressure within the heart accelerates cardiac action, even when all nerve branches, both of vagus and sympathetic, were severed. The muscular fibres are in bundles, the "primitive muscular bundles," consisting of the highest form of involuntary muscular fibre, and have cross-markings, or are "striped."

FIG. I.



Muscular Fibre of Heart (from Rindfleisch).

This mass of muscular fibre has a peculiarity in its blood supply, necessitated from its position and action. The coronary vessels, as the cardiac arteries are termed, spring from the base of the aortic column, from the dilatations, denominated the sinuses of Valsalva. They receive their blood supply, not from the ventricular systole, but from the

trophy. Hypertrophy gives it length, and dilatation width, while in simple dilatation there is a wide, diffused, but comparatively weak impulse. In chorea, and Basedow's disease, as well as nervous palpitation, the anterior chest wall vibrates over a space the size of the palm. In some cases of right-side dilatation and tricuspid regurgitation distinct pulsation may be seen to the right of the sternum at the second intercostal space, indicating the enlargement of the right auricle and its increased action.

Palpation.—Palpation is the name given to the application of the hand to the examination of the heart, and often furnishes valuable information in addition to that furnished by inspection. Thus palpation will corroborate the information which we have just seen is furnished by inspection, and it will not be necessary to repeat it. In a perfectly normal heart the finger may be placed on the exact apex beat, which can be felt distinctly. This admits of an instructive experiment, especially in a lean person. Having placed the finger tip on the apex beat, let the patient hold his breath. Then we find the apex beat becoming lost, until it is ultimately imperceptible behind the distended right ventricle. W. T. Gairdner calls the right ventricle here a water cushion. When the necessity for breathing comes this water cushion can be felt passing away until the apex beat is once more distinctly to be felt. This gives us a good idea of the temporary engorgement of the ventricles and their ready recovery, if not too frequent and long continued, when a permanent condition of dilatation is set up. In hypertrophy a blow-like stroke may be distinctly felt by the hand, and a considerable diffused heaving in hypertrophy with dilatation, while in simple dilatation a diffused and feeble slap may be felt, often with a struggling sensation, in the palpitation so often found with dilatation. In fatty degeneration no apex beat may be discernible, a significant fact, demonstrating that the heart is acting feebly; for there is something, almost indescribable by words, which divides the feeble, quiet, undemonstrative structurally diseased or atrophied heart, from the obvious struggling of a heart dilated but structurally sound. Palpation will also tell us of the "jogging motion" of Hope,

the embarrassed heart of Bouillaud in pericardial adhesion involving the costal pleura. Palpation gives, too, information as to disturbances of rythm, as to palpitation, irregularity, and intermittency, and in a dilated heart there is something pathognomonic in the few, rapid, feeble strokes preceding a halt, and then a sensation of "rolling over," as the contraction again commences with a comparatively powerful systole. Palpation, too, indicates cardiac aneurism of the left apex by the fulness and protrusion of the intercostal space over the left apex on every systole, as pointed out by Skoda, from the aneurismal portion being thrust forward.

From the application of the hand we also learn as to epigastric pulsation from dilatation of the right ventricle, where it is marked. There is also aortic pulsation, sometimes confounded with it, especially in the neurosal affections of the descending aorta. There is, too, a pulsation of the liver which is regarded by the Germans as of great diagnostic importance, as indicating tricuspid regurgitation. It is not certain how far it is due to venous regurgitation into the portal circulation, or to impulse communicated from the right ventricle.

The hand, too, will give further information; as, for instance, the friction of pericarditis becomes felt as well as heard, as a thrill, the "fremissement cataire" or "katzenschnurren," and aortic stenosis will often give a perceptible vibration or thrill. In dilatation of the aorta with roughening of the endarterium by atheroma a strong impulse with thrill can be felt by the finger in the manubrium sterni, especially when the patient holds his head forward; and thus, often a diagnosis can be made from aortic stenosis, whose murmur it simulates. Palpitation of the heart is sometimes complained of when another phenomenon is at work, viz., quivering of the intercostal muscles over the heart; and the patient may have distressing sensations, and refer them to the heart, when the hand will detect this muscular action quite distinct from the heart's action, which is steady and regular. B. W. Richardson in "Discourses on Practical Physic," gives an account of a false palpitation in an eminent man of science. Here "the palpitation was from some

muscular sphere from the gorged veins behind, until sufficient distension is produced to awaken the sleeping auricle and provoke contraction in it; this, in its turn, produces sufficient distension of the ventricle to provoke its contraction, and this passes on so swiftly as to create the impression of a synchronous contraction. The auricles, not possessing valves behind them, are not of any great muscular strength. From this absence of valves, a quantity of blood passes backwards at each contraction, an arrangement which, if it existed also in the ventricles, would make the heart of very little value as a propelling organ, it would be of about as much efficiency as a pump without valves, and that would not be much. When, then, the ventricle contracts, the backward flow into the auricles is, to a small extent no doubt, checked by the closed auricle, until the auriculo-ventricular valves are closed by the systole.

These auriculo-ventricular valves are not simple valves, like the semilunar valves, but have tendinous cords inserted into the ventricular surfaces; these tendinous cords terminate in muscular continuations, the musculi papillaries, which are inserted into the muscular walls of the heart, and contract with the ventricles. By this arrangement the valves are prevented from being washed backwards into the auricle by the ventricular systole, and are also drawn inwards, by the contractions of the muscular terminations of the tendinous cords. In the bird the valves are mere muscular folds, which are lifted by the in-rush of the blood, and then take part in the muscular contraction, forming, indeed, part of the chamber wall. This form is found in the lower mammals, or monotremnata, along with other bird characteristics. By the closure of these auriculo-ventricular valves, the blood is prevented from flowing backwards, and its progress through the arterial openings secured. Many experiments have been performed in order to test the perfection of these valves as to function, or, as it is called, their competency. The best known of these are the experiments of King, published in 1837. He found the mitral valve to be almost invariably competent, even under a heavy pressure exercised through the aorta, the aortic valves having been previously removed.

But he found the tricuspid not nearly so resistant, a weakness which it possesses in common with the rest of the right side of the heart, which is ever more prone to distension than the left side. From these experiments King evolved a theory of a normal competency in the tricuspid, now well-known as "the safety-valve action of the tricuspid:" an action supposed to protect the right heart from over distension and paralysis, and therefore very useful. But with all due respect to King's experiments, and his theory, this "safety-valve action" would simply mean a decided imperfection in the heart, which would render it practically useless, or nearly so. Unfortunately in practice one only sees too much of venous engorgement due to imperfect action of the heart, and if such an imperfection had been specially provided for us, it is somewhat difficult to see how the race could be maintained on the face of the earth. For tricuspid incompetency means not only venous engorgement, but also deprival of arterial blood to the system, and failure on the slightest call upon the heart.

We shall see further on, that tricuspid insufficiency is the most serious of valve failures, and is attended with the most deplorable consequences, and at no distant period, too. Though the question of tricuspid sufficiency or insufficiency is a matter of fact, and not of opinion, and therefore not to be affected by theories about it, still King's hypothesis has such a large circulation, that some remarks on it are not out of place. The writer considered the question at length in a paper in the "Edinburgh Medical Journal," for December, 1870, which may be referred to, and the conclusions will be briefly put here.

1stly. A certain amount of contraction has elapsed before the auriculo-ventricular valve is closed, by the backward flow of blood, and King never demonstrated that the tricuspid was then incompetent.

2ndly. "The moderator band of Reil"* is not inserted into the yielding wall, so as to admit more easily of the valve

* The reader will perhaps excuse the explanation that "the moderator band of Reil" passes from the interventricular septum to the yielding wall in the right ventricle, and is really merely one of the columnæ carneæ.

being rendered incompetent, but, in fact, the very reverse, being inserted at the point of insertion of the musculi papillaries of the outer-valve, or valve of the yielding wall, and thus tending to secure the apposition of the valves, even in great ventricular distension.

3rdly. That in the driving mammals, where such a provision would certainly seem indicated, all possibility of tricuspid incompetency is provided against by a most powerful right ventricle (in the dugong it is equal in size to the left, and the heart looks "double"), with strong columnæ carneæ, while the right auricle is also supported by strong tendinous trabeculæ. There is a provision for the requirements rendered necessary by their aquatic pursuits, in the shape of enormous sinuses, arterial in the cetacea and venous in the oceanic carnivora. The theory of King is simply untenable, and only tends to disguise the very serious nature of tricuspid insufficiency, as it is met with in practice. By these different valvular arrangements, the heart is converted from a mere muscular pulsatile sac into the beautiful, complex, steadily working machine we find it in its highest forms, with its muscular power directed and economised to the utmost by these very valvular folds; and any valvular incompetency at once reduces the human heart to the position of the lower cardiac types.

In addition to these beautiful arrangements for conserving the muscular force, the heart possesses a nervous supply which, so far as it is yet understood, seems of an exquisitely elaborate order.

The nerve supply of the heart is chiefly ganglionic, but nevertheless the fibres from the vagus have very important functions. For the elucidation of this innervation, it has been absolutely necessary to resort to experiment, and the rabbit, among other qualifications for this martyrdom to science, possesses a very exquisite cardiac innervation. The investigations made recently by Claude Bernard, Thirry, Cyon, von Bezold, Ludwig, and Professor Rutherford, have done much to elucidate this subject. The recent lectures by Professor Rutherford are not only the latest but the most perfect enunciation of what is known about this matter, and

his views, as found there,* will be used here to illustrate this difficult subject. The great nerve supply of the heart consists of ganglia, lying chiefly in the sulci betwixt the ventricles and auricles, which are part of the great sympathetic, and, as such, are in connection with the medulla oblongata. By these the sensation of distension of the muscular sphere is received, and the notice to contract evolved. But irregular contraction of the heart's fibres would be inconvenient, and thus a regulating force comes in from the fibres of the vagus. These co-ordinate the nerve force evolved by the cardiac ganglia and the distension, so that the nerve force evolved does not produce contraction before the chamber is filled, neither does irregular contraction from irregular distension, that is the distension is not equal on every fibre at first, result, but a uniform synchronous contraction of all the fibres. The reasons for this opinion are these: 1. The heart can beat rythmically when cut out of the body in some animals, and thus a nerve influence must be evolved rythmically by the cardiac ganglia. 2. Galvanizing the vagus will delay, and, if strong, arrest the heart's contractions; thus the vagus possesses a retarding, co-ordinating action, called "inhibitory." 3. Increased pressure on the heart from within will induce more frequent contraction, even when all cardiac nerves have been severed. It would thus appear that the sensation of distension is somewhat antagonistic to, and balanced by, the inhibitory action of the vagus, and in ordinary working the sensation of distension and the evolution of the nerve order to contract by the ganglia are controlled by the vagus.

But in addition to this innervation come the vaso-motor and vaso-inhibitory nerves, of which a brief and, if possible, lucid explanation will be attempted. The vaso-motor nerves are spread all over the blood-vessels, and are part of the ganglionic system. The arteries in a white-eared rabbit can be seen to alter their calibre, and that somewhat rythmically, and changes in the calibre of veins have been observed. Section of the cervical sympathetic in the neck, it is well

* See "Lancet" for 1871—1872.

known, leads to paralysis and distension of the vessels of the head, *i.e.*, their semi-contracted normal condition cannot be maintained after section of the sympathetic trunk, with which their vaso-motor nerves are connected. These vaso-motor nerves seem to possess a vaso-motor centre lying betwixt the corpora quadrigemina and the calamus, and irritation here will produce vaso-motor spasm generally, and, from the opposition offered thus to the blood stream, excited action of the heart and palpitation. The vaso-motor nerves have also inhibitory nerves, *i.e.*, nerves which counteract the vaso-motor nerves and relax the blood-vessels by suspending the action of these vaso-motor nerves. The inhibitory nerves are connected with the vagus, and in the rabbit this section forms the superior cardiac nerve. This nerve seems also the "sensory nerve" of the heart. Certain nerve fibres of the heart pass to the endocardium and record over-distension. Cyon found that by passing a stream of serum through the heart so arranged as to be able to add carbonic acid, to the serum, and cut if off again at will, that the carbonic acid paralysed the heart, and its contractions ceased, but were resumed when the carbonic acid was cut off. This all tends to show us something of the beautiful nerve arrangements by which the circulation is regulated and balanced. The heart becomes distended to a perceptible degree, and its sensory nerve being also the vaso-inhibitory nerve, this distension leads to the vaso-inhibitory action being put in force, and the vessels of the peripheral circulation become dilated. This at once leads to diminished arterial pressure, being equal to so much increase in the arterial system in capacity to hold blood, the ventricles are relieved by this, and the distension of the heart is remedied. Not only does this occur, and exercise an immediately relieving effect, but this vaso-motor inhibition extends to the vessels of the coronary circulation, and thus a freer blood supply is furnished to the heart walls, and hypertrophy, giving greater power to withstand distension, which is equivalent to being able to more completely contract and empty themselves, results. The correct appreciation of this double innervation of both heart and blood-vessels will be of great importance in aiding a more thorough understanding

of various morbid actions, as well as a more perfect comprehension of the causation of hypertrophy.

The two muscular ends of the circulation, the central accumulation into one mass, the heart, and the general peripheral distribution, the arterioles, are thus balanced in an exquisite manner, and maintain a blood equilibrium, by acting and reacting in a manner which must excite the admiration of all, while this mutual reaction can so act and react as long to avert disturbance of balance in the circulation—a subject the importance of which will become more and more apparent as different diseased conditions come under our notice.

CHAPTER II.

THE HEART'S POSITION AND MODE OF EXAMINING IT—INSPECTION — PALPATION — PERCUSSION — AUSCULTATION — AID DERIVED FROM ARTERIAL AND VENOUS SYSTEMS—VOCAL RESONANCE.

THE position of the heart in the thorax may be rudely described as extending from the second right intercostal space, under which is the right auricle, to the fifth left intercostal space extending as far to the left as the nipple, where the left apex may be felt. It alters somewhat with the position of the body, and with deep inspiration and expiration, so far as its limited bounds will permit. Enlargement gives it a tendency to fall down somewhat, probably simply from its increased weight. Placed thus in a comparatively fixed position, certain relations to other points are so fixed that we can refer to them in speaking of the heart, as, for instance, its apex beat being in the fifth intercostal space, the sound of the aortic valves being heard in its maximum intensity at the articulation of the right second costal cartilage, the pulmonary sounds at the third left costal cartilage, and the tricuspid at the ensiform cartilage; or again, that the left auricle lies behind the third rib and the second and third intercostal spaces. Occupying such a position in the thorax, it admits of being examined by inspection, palpation, percussion, and auscultation.

Inspection.—Inspection tells us how far the patient's physique is good; how far the thorax is well formed; whether the intercostal spaces are widened or narrowed by inspiration; whether each side of the chest is playing well, and the movements of respiration symmetrical. In making this inspection a good light is required to give more than mere negative information as to the heart itself; consequently the patient should face the light, a position which will also put the medical man's face in the shade, a

matter often of some importance. When the light thus falls on the thorax a faint movement may be seen at the fifth intercostal space, synchronous with each radial pulse. In very thin persons, especially if under excitement, this movement may extend over a space the size of the palm of the hand.

Inspection will also give us information as to various morbid or abnormal conditions, as follows:—In many cases of heart disease, especially where there is hypertrophy, there is a protrusion of the thoracic wall over the region of the heart. This is very common in young persons before the costal cartilages are set. We also can see at other times an epigastric fulness from depression of the diaphragm due to increased weight within the pericardial sac from effusion or enlargement of the heart itself. The intercostal spaces may be seen bulging in pericardial effusion, while a retraction may be noticed when pericardial adhesion has glued the heart to the costal pleura of the anterior chest wall. Another, not so well known consequence of pericarditis, viz., paralysis of the diaphragm from the inflammation affecting the phrenic nerves, may be detected by inspection. Here in inspiration there is not normal fulness but retraction, and on expiration not retraction but protrusion; the diaphragm no longer taking part in the respiratory movements, but merely being affected by them.

As to the heart itself, inspection will give us information often as to its position; as, for instance, when enlarged, it is apt to fall a little, or the apex beat to be lower from the elongation of hypertrophy. The heart, too, may be displaced to the right side by left side pleurisy, by retraction of the right lung, mediastinal tumour, or enlargement of the left lobe of the liver. It may be too much to the left from right side pleurisy, left side lung-retraction, and frequently from enlargement of the liver. The heart's action is also, to some extent, recognisable by inspection, and in fatty degeneration of the heart itself, in atrophy, and in pericardial effusion, the apex beat is invisible. In hypertrophy there is a forcible blow, especially when there is some dilatation with great hypertrophy, as in aortic regurgitation and in right-side hyper-

aortic recoil or aortic systole. The explanation of this is obvious, for were the coronary circulation to receive its blood supply from the ventricular systole, it would receive that blood supply at the most unfavourable time, namely, the period of muscular activity, when the muscular contraction would oppose the entrance of the blood. Instead of this it receives its blood supply from the aortic recoil, and during its own diastole, or period of muscular flaccidity. For a proper recognition of this fact and its bearing, as we shall see, on a very common valvular lesion, aortic regurgitation, we are indebted to Mauriac. The elastic aorta is distended by the ventricular contraction and recoils by virtue of its own elasticity; the flow of blood backward into the ventricle is arrested by the closure of the aortic semilunar valves, and the blood passes into the coronary vessels from the aortic pouches of Valsalva. This is one of the peculiarities in the cardiac circulation of great importance to the continuation of its action. Another is the manner in which the coronary veins open into the venous circulation. This is effected by their passing obliquely through the wall of the right auricle, forming the Foramina Thebesii. By this oblique perforation regurgitation is, to a very great extent and normally, completely prevented. The same arrangement exists in the mode by which regurgitation into the ureters from a distended bladder is prevented. This peculiarity was pointed out by Wardrop, who showed its importance in preventing the heart from being paralysed by a reflux of venous blood, charged with carbonic acid, when already suffering from right-side distension or engorgement. For not only would the carbonic acid have acted as a direct paralysing agent, but the venous blood gorging the coronary veins, would obstruct the flow of arterial blood coming in by the coronary arteries. The muscular contraction of the heart compresses all the smaller vessels within its structure, and empties their contents into the coronary veins, which, in their turn, convey the blood into the right auricle during the auricular diastole, which commences ere the ventricular systole is over. The mode by which the veins perforate, secures the cardiac circulation against venous regurgitation, and when the muscles

become flaccid behind the aortic valves, the blood in the aortic systole finds the cardiac circulation empty ready for the flow of blood into its relaxed muscular structure.

During this diastole the heart-structure is not only being supplied with pabulum, and its integrity maintained by nutrition from a free vascular supply, but also it is now enjoying its brief, fitful sleep. In its incessant round of labour no long repose is compatible with the continuation of the existence of the organism. It must not, however, be supposed that the actual amount of sleep enjoyed by the heart is small; that would be incompatible with its active function. The diastole is much longer than the systole, and Marey, from cardiographic tracings, calculated the systole to occupy no more than one-fifth of each cardiac revolution, while the remaining four-fifths, the diastole, are devoted to sleep and to feeding.

From Professor Traube's tracings, with his new improved cardiograph, the writer feels inclined to think this calculation of Marey's very nearly correct. The heart then, at a rough estimate, sleeps no less than 19 out of the 24 hours. But the auricles sleep even more, and their thin walls have comparatively long periods of rest. The thicker ventricles have a longer period of activity during each cardiac revolution.

This calculation of Marey's is formed from a normal and slow pulse. When the pulse rises, there are so many more contractions, or systoles, each minute, and as this increase of frequency takes place at the expense of the diastole, *i.e.*, the duration of each systole is but little, if at all, affected by increased rapidity in the ventricular contractions, and consequently the heart's sleep is infringed upon. Increase in the rapidity of the pulse tends to exhaust the heart by diminishing not only its sleep, but equally the time during which it is receiving its nutrition. Thus the difference betwixt a pulse of 72 and a pulse of 144 is, that in the one case the heart sleeps and feeds four-fifths of its time and works one-fifth, and in the other the sleeping time is reduced to three-fifths, while the working time is two-fifths. This is a very serious difference in an organ whose labour is so incessant.

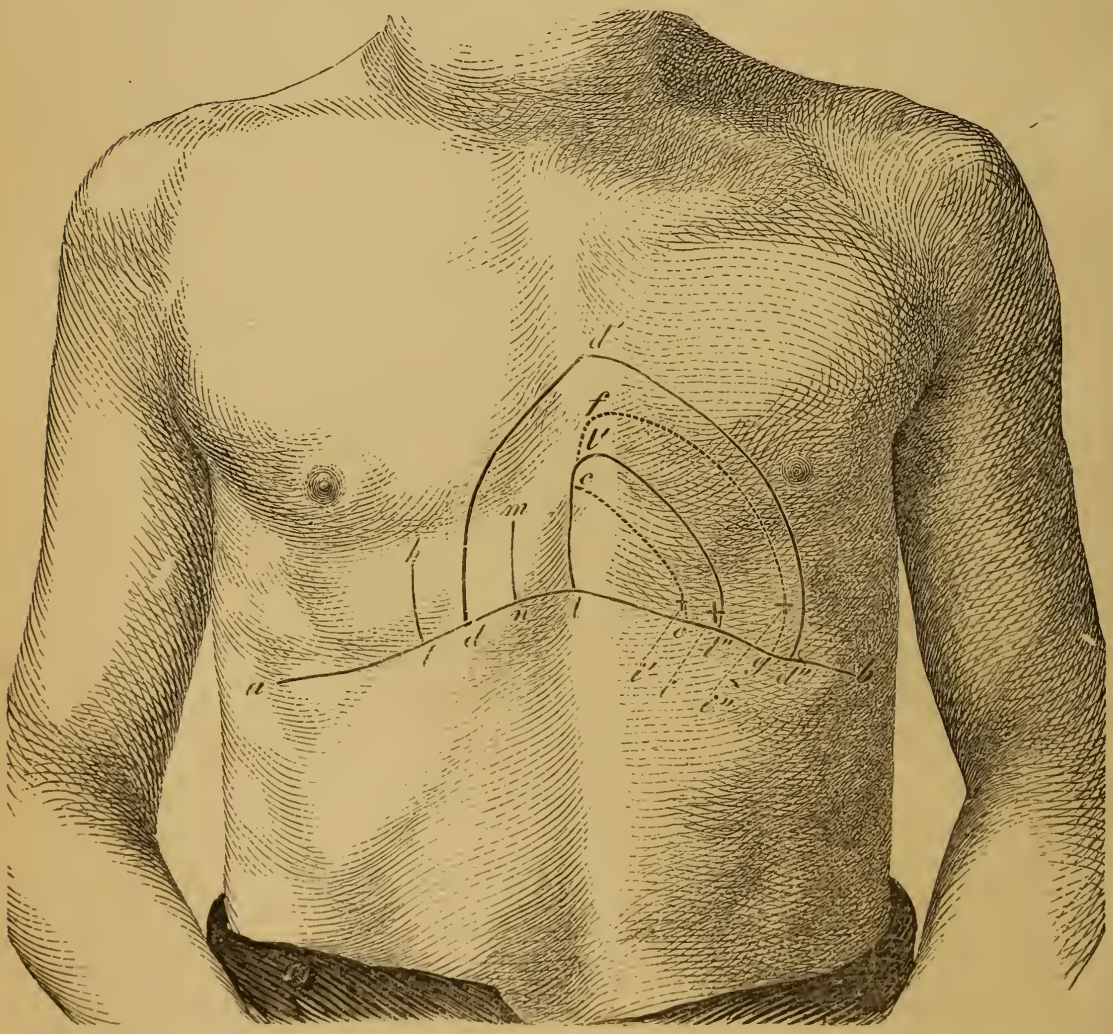
During this brief sleep, the blood is pouring into the flaccid

pulsating action immediately below the heart; it was epigastric, and had no relation to the heart."

Percussion.—This is one of the most important means of ascertaining the condition of the heart, so far as its size and shape at least are concerned. But percussion is surrounded by many difficulties and sources of fallacy, and requires the greatest care and circumspection in its performance. A small portion of the heart is alone in direct contact with the chest wall forming a rudely triangular space, varying in size on inspiration and expiration. A deep inspiration will so inflate the lungs as to bring down the thin edge of the lungs further over the heart, while a full expiration permits more of the heart to come in contact with the chest wall. It follows from this that the lungs may interfere very much with the area of cardiac dulness by increasing it in cirrhosis of the lung, for instance, and diminishing it in emphysema, or when the left lung is turned down over the heart by an adhesion to the costal pleura. From this area of complete dulness the lung interferes and grows rapidly thicker, so that the outline of the heart itself is well covered by lung, and gives only deep-seated dulness. Practice and patience will alone endow a man with power to estimate, fairly correctly, the outline of the heart by percussion, and by no other means can it be done at all. The knowledge is well worth the trouble required to attain it. In order, then, to learn something really valuable, as this information is, viz., how to map out a heart, and also how to learn that very useful auxiliary to percussion, the sense of resistance, perfectly indescribable by words, I can recommend to the beginner the following plan :—Take patients with presumably healthy hearts, and with a thorax not too much covered with either muscle or fat, and first ascertain the exact apex-beat with the finger tip. Then draw on the skin a heart about the size of a closed fist, making the right auricle come up to the second right costal cartilage, which will give about the correct position and size of the normal heart. Thus the student will have a guide to the interpretation both of the sound elicited and the sensation of resistance offered. The gradually more deep-seated dulness of the upper border will be better ap-

praised, while the outline of the lower border, in close apposition to the liver, will be more clearly distinguished from the liver-dulness immediately beneath it, for the heart is merely separated from the liver by the diaphragm and some serous layers of no great thickness. If this plan is not satisfactory to the reader it is to be regretted, but really I can communicate no secret for a more easy method of acquiring a solid knowledge of percussion in defining the heart. In order to assist him the following plate is copied from von Dusch's, the most recent German book:—

Fig. II.



Outline of Heart-Percussion-Dulness, of Complete Dulness, and of Apex-beat (in the recumbent posture on each side).

a b, line of diaphragm; *d d' d''*, outline of heart-dulness when lying on the back; *h l*, limit of dulness when on the

right side; *m n*, ditto on left side; *l l' l''*, limit of complete dulness when on the back; *l f g*, ditto when on the left side; *l c e*, ditto when on the right side; *i*, point of apex-beat when on the back; *i'*, ditto when on the right side; *i''*, ditto when on the left side.

This will aid much in attempting to learn percussion, the value of which increases much with the increasing skill of the percussor. When sitting or standing, the heart will be a trifle lower than when in the recumbent posture. Having become fairly acquainted with the normal heart, as indicated by percussion, the difference in size and shape in it will not present so much difficulty in their recognition. Thus in left-side hypertrophy the dulness will extend downwards, without great increase in width, and more to the left, merely an extension of the left ventricle outwards and downwards. Right-side enlargement will increase the boundary of the lower edge and extend it over the 3rd, 4th, and 5th right costal cartilages. General dilatation of the heart is rather globular than pointed, while pericardial effusion is pyramidal, with the base downwards, and becomes broader in the horizontal posture, and narrower when upright.

It will now be necessary to point out some of the sources of fallacy, the different circumstances which interfere with the indications of percussion, and lead to error. A variety of different conditions militate against the absolute accuracy of the information derived from percussion, and a brief *resumé* of them may be of service.

Firstly, as to the different morbid conditions which will be liable to mislead us by increasing the apparent side of the heart. Retraction of the lung by bringing a greater portion of the heart in close contact with the chest, will apparently add to the heart's dulness, but that will only extend to the area of complete dulness. Consolidation of the lung by pneumonia, or tuberculosis, will add to the heart's apparent size. In addition to these are pericardial effusions and thickenings, aneurism of the ascending aorta, substernal abscess, tumours in the anterior mediastrium, simple or malignant, a thymus gland still remaining, pleuritic effusion, more so if limited by adhesions, &c. Fat or thick muscles

will, on the outside of the chest, more or less interfere with the percussion; and in females the left breast is sometimes in the way.

The heart-dulness is lessened by atrophy of the heart itself, or congenital smallness, by an emphysematous condition of the lung, or by the lung being bound down over the heart by a pleuritic adhesion. Deep inspiration will reduce the area of complete dulness, while change of position will alter the limits somewhat. In pneumopericardium the heart's area becomes tympanitic.

When taken along with the information furnished by inspection and palpation, percussion becomes a valuable addition to our means of examining the heart, and its sources of fallacy will be fairly eliminated in the great majority of cases by the aids derived from the other measures, and even more by the information furnished by the remaining means of examining the heart, viz., auscultation, a method practised immediately, for some time before Laennec added so much to its importance by the use of a mediate agent, the stethoscope.

Auscultation.—This last is the most beautiful and most perfect means we possess for ascertaining the condition of the heart. By it we can read more exactly the conditions of enlargement with which we have been made acquainted by the previous measures; we can interpret how much dilatation exists, and with how much hypertrophy combined; how far the heart is equal to its work by the perfection of its rythm; how its valves are working; if obstruction exists; if regurgitation be present. We can ascertain very often on what that enlargement depends; we can feel, as it were, the structural condition of the heart itself. But to enable us to do all this by auscultation, we must first make ourselves certain, as far as we can, of the sounds in health, their modes of origin, and the conditions which alter them. The heart's sounds are two, the first and second sound, so-called from the order of time in which they occur in the cardiac revolution. The first is the systolic sound, accompanying the ventricular contraction; the second is diastolic, or heard during ventricular flaccidity. Betwixt these two is a pause, very short, but still distinct. After the second sound, and before

the next systolic sound, comes a longer pause. The pauses and sounds are nearly proportioned to each other, the long pause precedes the long first sound; the short pause precedes the short second sound. Supposing the dotted lines to represent the pauses, and the unbroken lines the sounds, we find each cardiac revolution to consist of ------ —— -- —. In order to thoroughly comprehend this, we must just go shortly over the heart's action, which is to deliver its contents into the arteries. Thus during the long pause, or period of rest, the blood is rushing from the veins into the heart's chambers inaudibly; nor is the normal auricular contraction audible. On systole or ventricular contraction we have the first or long sound produced; a short pause, and then the recoil of the elastic arteries drives together the semilunar valves, causing the short, sharp, second sound of valve closure. This is the cardiac revolution, and now we must see how the first sound is produced.

The production of the first sound has been the subject of an immense amount of discussion, which it would be needless to review here. It is now generally admitted that the first sound has two factors, the chief factors, and other smaller auxiliaries. The two chief factors are muscular contractions of the walls, and closure of the auriculo-ventricular valves. In order to get a good conception of the character of muscular sound, the student can first apply the stethoscope over the heart. Take the character of the sound, and then apply the stethoscope over the ball of the thumb, and put the muscles of the thumb into action rythmically. This can be done without any movement of the skin and friction on the stethoscope, which would be a source of error.

A little of such practice will soon enable the young auscultator to distinguish for himself the character of muscular sound more perfectly and clearly than any amount of reading could teach him. Having learned to distinguish the character of muscular sound, he will soon be able to distinguish the other chief factor, the sound produced by the flapping together of the auriculo-ventricular valves. This is a "slapping" sort of sound, approaching the character of the second sound. In hypertrophy of the muscular walls, the muscular sound is

often predominant, giving a dull, muffled sound, while at other times, as in dilatation, the thin wall emits but a thin muscular sound, and the flapping together of the auriculo-ventricular valves is the predominant sound. In hypertrophy with dilatation sometimes the valves are so forcibly driven together as to produce a "clanging" or "ringing" sound. The sounds are altered by thickening of the free edges of these valves, or by great thinness. In addition to these two chief factors must be added a third cause of sound, viz., the blow of the heart's apex against the chest wall. This is attributed to the recoil of the heart on expelling its contents, like the rebound of a musket, but it seems more probably to be due to the elongation of the arteries, and especially of the aorta, on distension by the ventricular contraction. In addition to these the Germans attach much importance to the rushing of the blood through the aortic and pulmonic orifices, as a factor in the production of the first sound.

These sounds go to make up the first sound, and may be found with one or other preponderating. The blow of the apex against the thoracic wall is often very apparent, from the impulse communicated to the stethoscope, more so indeed than to any impression it makes on the internal ear, as the other causes of sound will generally be increased along with it. The first sound may become almost entirely lost in fatty degeneration, or may approach the second sound in character.

The second sound is now attributed entirely to the closure of the semilunar valves. The sounds of the aortic valves are stronger and thicker than the pulmonary valve-sounds, and can be distinguished from the pulmonary sounds. The aortic sounds can be most distinctly heard at the articulation of the second right costal cartilage with the sternum; at this point the aorta comes nearest to the anterior chest wall from behind the pulmonary artery. The pulmonary sound is best heard over the third left costo-sternal articulation, and is only heard at this point very commonly. These sounds are normally heard over very limited areas, and may with a little care be readily distinguished: the aortic sound fuller and stronger, the pulmonary sound sharp and clear. Obstruction to the blood-flow accentuates these sounds, and

this accentuation is often of great diagnostic value. In Bright's disease the aortic sound is ever increased, and any pressure or obstruction in the aorta itself will cause it. But it is in the pulmonary second sound that the accentuation is diagnostically most important; all obstruction to the pulmonic circulation, as in extensive lung disease, congestion, whether simple or arising from left side incompetency, becomes audible by accentuation of the second sound. Skoda first pointed this out, and showed its importance as a means of estimating the amount of mitral disease. Accentuation of the pulmonary second sound is pathognomonic of stress on the right ventricle, however produced. It is almost always an indication of enlargement of the right side of the heart. As we proceed to the discussion of various morbid conditions, the reader will see the great importance of alteration of the second sound, and the diagnostic importance of his being able to distinguish any variation in the character of this sound.

Murmurs.—The alterations in the sounds of the heart are all-important, but diagnostically their being supposited, or merely marked by a murmur, is of the greatest moment. Murmurs are differently produced, and are of two kinds, the obstructive and the regurgitant. The obstructive murmur is produced by the blood being forced over a roughened orifice, or a narrowed orifice. It is thus of a rasping or sawing character, and often not unlike a fine saw going through a piece of soft wood. The obstructive murmur is usually significant of stenosis, and may be found at the arterial orifices, and at the mitral valve not uncommonly, but tricuspid stenosis is practically unknown. The regurgitant murmur is produced by reflux, or regurgitation through a valve which is no longer closing perfectly, and cutting off the backward flow of blood. It is a soft murmur as compared to an obstructive murmur, and is usually of a blowing sound, especially in auriculo-ventricular regurgitation. It may be found at any of the four cardiac orifices, by far the most rarely at the pulmonic orifice. Sometimes the valve becomes both narrowed and incompetent, and then we have a double murmur consisting of both obstructive and regurgitant murmurs, the regurgitant following the obstructive. Murmurs oc-

curring during ventricular contraction are termed systolic; during ventricular rest they are termed diastolic, and are connected with imperfectly arrested backward flow on arterial recoil.

Thus we find that systolic murmurs are four; diastolic two. The four systolic murmurs are of two classes, obstructive and regurgitant. During the ventricular systole murmurs may arise either at the arterial orifices, obstructive murmurs, or from auriculo-ventricular insufficiency, regurgitant murmurs. These murmurs are all isochronous. It is obvious then that other means must be invoked to determine which of these four a systolic murmur may be. These are two: 1. Character of sound, not very trustworthy at all times; and 2. Position of maximun intensity, of very considerable aid in diagnosis. The obstructive murmur is harsher, usually more pronounced, and heard over a greater area. The regurgitant murmur is softer, more blowing, and heard over a more limited area.

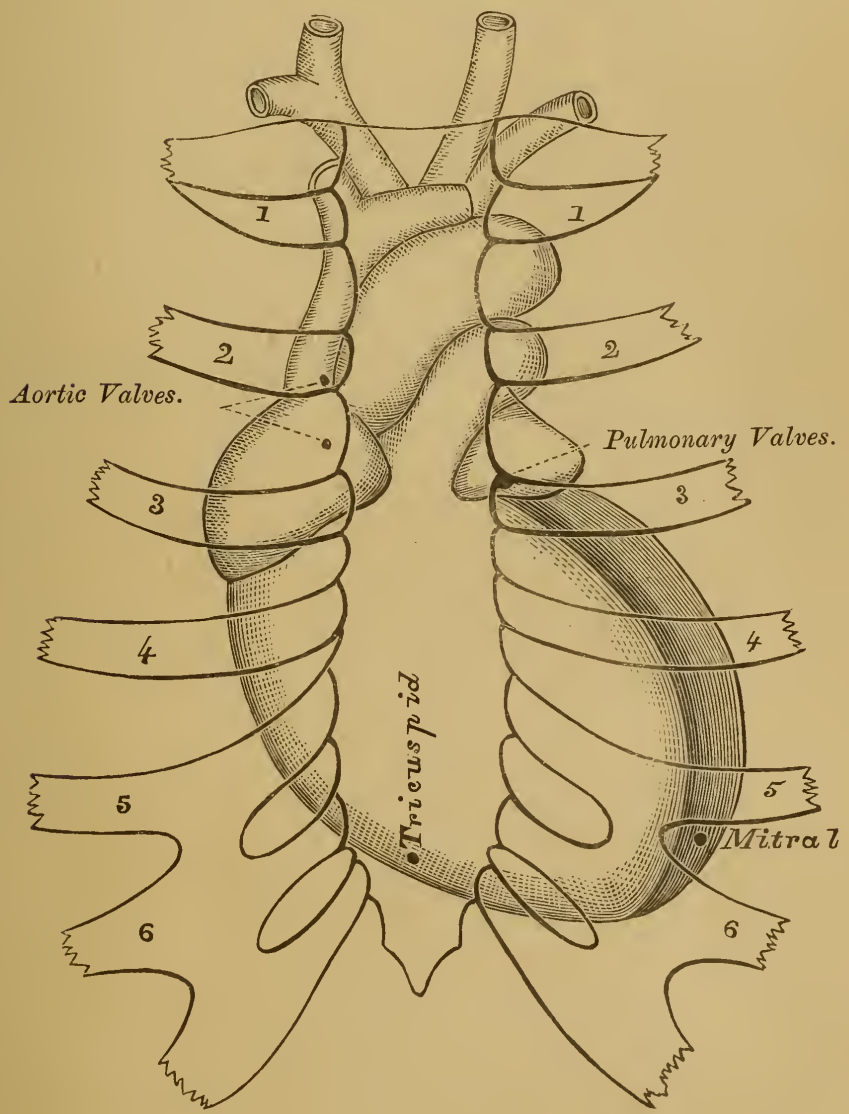
But in so speaking, it must be understood that the typical murmur is being here described. For murmurs run into each other, exist together, may be heard over greater or lesser areas, are congenital, intermittent, and are at times in various ways perplexing. But ordinarily they may be as fairly relied upon as any other diagnostic means we possess.

Then as to position, it is obvious that each valve may have two distinct forms of disease, and consequently two murmurs, which may occur separately or combined. But the period at which heard, prevents this from being confusing. Each orifice has then an area over which its murmurs are heard with greatest intensity. As a crown piece would cover the four valves of the heart, it is obvious that position of maximum intensity would be rather uninstructive and confusing, than tending to throw light on the subject, if it were not that each murmur radiates a little outwards, according to what the late Hyde Salter called "conduction" and "convection." This radiation makes position of maximum intensity a point of great importance in estimating which of the four systolic murmurs we have got to deal with.

Thus an aortic systolic murmur is heard a little way from the aortic valves, and is most distinct where the aorta curves

forward towards the sternum, *i.e.*, the second right costo-sternal articulation. The pulmonary systolic murmur is heard very closely over the valves, as the artery soon curves from the sternum, and is in its maximum intensity at the third left costo-sternal articulation, and usually only heard at this point. A systolic murmur heard in its greatest intensity towards the left apex, and even further to the left, an inch even behind the nipple sometimes, is mitral regurgitant. It is often heard over a much larger area, but that is the area of its maximum intensity, and commonly it is confined to that area. A tricuspid murmur is only heard, and that too faintly,

Fig. III.



over the ensiform cartilage. These different areas are well marked and illustrated by the accompanying plate.*

These four murmurs then, can be arranged according to these different means of distinguishing them, so as to make their distinction not very difficult.

Valve.	Murmer.	Time.	Character.	Point of maximum intensity.
Mitral.	Regurgitant.	Systolic.	Blowing.	Left apex, to the left.
Tricuspid.	Ditto.	Ditto.	Ditto.	Limited to ensiform cartilage.
Aortic.	Obstructive.	Ditto.	Rasping.	Second right costo-sternal articulation.
Pulmonary.	Ditto.	Ditto.	Ditto (fine).	Third left costo-sternal articulation.

These are the systolic murmurs which mark, or obliterate the first sound; they are more or less common, the mitral and aortic most frequently met with, and then the tricuspid, the pulmonary being very rare. It is obvious that the relative frequency has nothing whatever to do with the diagnosis, but it has sometimes a value, especially in guiding a tyro, who usually finds more rare murmurs during the earlier part of his diagnostic education than he does later on, when his diagnosis is more perfect.

Lesions heard during the time of the heart's second sound are necessarily regurgitant through the semilunar valves, and may be either aortic regurgitant, very common, or pulmonary regurgitant, very rare. The regurgitant murmur is always heard over the base of the heart, and often also towards the apex, and extends over the third, fourth, and fifth costal cartilages and corresponding portion of sternum. The aortic regurgitant murmur is then diastolic, and that almost establishes its position, and renders its recognition almost independent of the character of the sound (blowing,

* This plate is taken from Da Costa's well known work on "Medical Diagnosis," with a slight alteration to meet the writer's views as to the position of the pulmonary valve-sound. Da Costa places it over the second left costo-sternal articulation, while the writer agrees with W. T. Gairdiner in placing it over the third left costal cartilage.

or perhaps harsher), and of its area. The diagnosis of a pulmonic murmur from an aortic murmur when regurgitant is difficult, and it is fortunate that practically the question resolves itself into one of relative frequency. (For the requirements of diagnosis here, the reader must look to the chapter on Valvular Lesions.)

During the time of ventricular inactivity, two other murmurs are heard, viz., mitral and tricuspid obstructive murmurs. These were once described as diastolic murmurs, and we are indebted to W. T. Gairdiner for first clearly pointing out that they are not diastolic truly, but, in fact, presystolic murmurs. That is, they are caused by the presystolic auricular construction, and run into the first sound. Here, then, are theoretically two murmurs, but practically only one, for tricuspid obstruction has not yet been satisfactorily diagnosed in life, and in two cases occurring to such accomplished diagnosticians as Bamberger, and W. T. Gairdiner, in each case there was a tumour pressing on the right auricle, and not a tricuspid stenosis, as supposed.

The mitral obstructive murmur as heard over the left apex, is presystolic, sawing, and not to be confounded with any other murmur practically. Thus really the number of murmurs is not so very confusing for the beginner, and most murmurs are single. The difficulties are not so great as to be discouraging, and a little careful attention will usually suffice to read this language of murmurs, and in a little time the student will be able to interpret pretty correctly the double or combined murmurs, whether both obstructive and regurgitant at one orifice, or the two murmurs belong to two separate orifices. Some murmurs, depending on curious congenital malformations, and on aneurism of the ascending aorta, or when a diseased valve is so associated, will tax the diagnostic skill of any one, from the redoubtable Skoda downwards, and the beginner should not be deterred by meeting with such a case early on. Neither should he lose faith in murmurs from finding no lesion after a very decided murmur, for Skoda has pointed out the origin of murmurs in irregular action of the musculi papillaries, where, naturally enough, no lesions are found. Lesions, too, are sometimes

found which gave no sign during life, and this is especially the case with lesions of the left side when tricuspid failure co-exists. It seems here, too, that the small amount of blood propelled through the heart is insufficient to cause a murmur at a narrowed orifice.

Murmurs, too, will intermit and be very perplexing, and even an aortic regurgitant, the most constant of all, will sometimes become inaudible.

Anœmic murmurs are usually aortic, but may be mitral. Peacock says that anœmia will often magnify an organic murmur in a rather perplexing manner. On the other hand, murmurs will often become audible when the heart's action is improved by treatment, which were previously inaudible, and as the heart sounds become more distinct, murmurs will often become audible as well.

A murmur resembling a musical note is sometimes found, and is supposed to be due to a shred of lymph hanging from the free edge of a valve and vibrating in the blood current.

Aid derived from Arterial System.—This will often afford us useful information, as the elastic arterial system, while healthy, gives out very accurately the impression made upon it by the heart. It is somewhat customary, too, to feel a patient's pulse early in his examination, and before physically examining his chest, and therefore the indications derived from the arterial system are very useful, and may give us the direction, at least, in which to conduct our examination. Its frequency generally first attracts our attention, and the counting of the pulse by the watch was the first attempt to aid our diagnosis by an instrument. This mere frequency is often suggestive, especially if it be found to remain, and not be merely the result of nervousness on the part of the patient. Great frequency is not a pleasant symptom, while unusual slowness is usually neurosal. But the frequency is of small moment usually as compared to its strength or force. This is usually in proportion to the ventricular impulse, and at once establishes a step towards the diagnosis. Thus it is full and strong in hypertrophy and weak in dilatation and fatty degeneration. Rythm is very

important, when good, giving a fair presumption of power in the heart; when irregular and even more intermittent it is of bad omen, but this may occasionally be neurosal. Sometimes a pulse is delayed, especially in elderly people, and the radial impulse is distinctly behind, and not synchronous with the ventricular systole. Then a pulse will sometimes be "full," but not hard and incompressible. This indicates vaso-motor inhibition and a dilated state of the vessels; the opposite condition of small, "wiry," incompressible pulse is connected with vaso-motor action and a contracted state of the vessels, very marked in hysterical palpitation and in abdominal inflammations. At times the real rate of pulse is somewhat masked, and it will give an apparent rate, not borne out by counting it; thus there is a quick slow pulse, *i.e.*, appearing quicker than it is, and a slow quick pulse, *i.e.*, being quicker than it appears to be. Then a pulse may be hard and incompressible, that is, pressure with the finger cannot obliterate it; here there is usually left side hypertrophy; at other times, in cardiac debility, or in anœmia, the pulse at the wrist is easily obliterated. It must be borne in mind that atheroma magnifies the ventricular impulse, and gives a heave to the pulse; this is well seen in the temporal artery when, sinuous and rigid, it is distended by ventricular systole and becomes widened and elongated. Of course atheroma is usually accompanied, in the earlier stages at least, by hypertrophy of the left ventricle, and thus the ventricular systole is strong and sustained. This combined condition is often misleading in conditions of temporary sickness. In a near relative of the writer's this condition existed, and even during physical depression, even when tonics and stimulants were really needed, the strong radial pulse from a hypertrophied left ventricle and atheromatous arteries almost seemed to indicate venesection. At times of greater depression, as after acute diarrhœa, a distinct intermission could be felt, apparently quite out of place in such a pulse, but really indicating correctly the passing condition. This effect of atheroma, or the impression created by a pulse, is well worth noting, and will often be found useful in practice.

The pulse is often very significant of a valvular lesion, and will sometimes be sufficiently marked to aid much in diagnosis; thus, in aortic obstruction it is small, but sustained, from the hypertrophy which accompanies such obstruction. In aortic regurgitation it is "splashing," or "like balls of blood shot under the finger," the arterial distension being great from the hypertrophied and dilated ventricle, but unsustained, by reason of the failure in the semilunar valves; in mitral obstruction it is small and regular, not sustained, as in aortic obstruction, while in mitral regurgitation it is irregular in volume, that is, the pulse varies in volume markedly, one beat being full, and the next small, depending upon the varying amount of blood which passes back through the imperfect mitral valve at each systole. This must not be confounded with the irregularity in time, the unrythmical pulse of dilatation, though both may occur together. In right side disease the pulse is ever small, and when right side failure co-exists, the pulse is less correctly significant of each left side condition, and possesses a general character of smallness. The right side really empties at every stroke so much of the blood in the veins through the lungs into the left heart, which transmits it more powerfully on into the arteries. But though the left side is so strong, it cannot affect the bulk of blood passed over to it by the right side; any defect in the original lifting pump is felt all over the distribution of the vessels, and a leak in that pump affects the volume in the most distant distribution.

The radial pulse will often alone tell us that in a heaving, strongly-acting heart, with a small pulse, the hypertrophy and action is in the right ventricle, and not the left; for equal action in the left would not be without effect on the radial pulse. This right-side hypertrophy is compensatory to some morbid action lying betwixt it and the radial pulse, and is neutralized thereby, as by some disease of the lungs, pulmonic circulation or left heart. The pulse, too, is affected by anœmia, and when anœmia is present, the pulse cannot faithfully reflect the impulse of the heart.

The arterial system will also convey murmurs, and the murmur of aortic obstruction may be distinguished in the

carotids, brachial, and femoral arteries. Murmurs will be created in arteries by pressure on them by a tumour, or the stethoscope itself, and every arterial murmur is not indicative of aneurism, when not cardial, but may depend on other causes, or be created by the mode of examination. Arteries when numerous possess a souffle, as heard in the placental or utero-placental souffle, in an enlarged thyroid gland, by dilated arteries, in Grave's, or Basedow's disease. The wide dilated aorta often found with atheroma has a murmur, caused by its roughened atheromatous interior, and this systolic murmur may not only be readily heard, but felt by a finger on the manubrium sterni. Here, especially when the patient thrusts his head forward, a thrill can be distinctly felt, along with the huge, long heave of the dilated, thickened vessel.

A subclavian murmur is often heard, especially in labouring men, which may be aneurismal, but is more often due to tubercular infiltration of the apex of the lung, or, as B. W. Richardson thinks, due sometimes to pressure of the subclavius muscle.

Aid derived from the Venous System.—The venous system furnishes us also with valuable information, even to the first sight in the cyanotic condition of venous congestion. In failure of the heart, especially when the right side is yielding, the veins are too full of blood, or, as the Germans say, the blood is lying too much on the venous side. Betwixt the two muscular terminations of the system the blood lies, and in health a balance is maintained by means of mutual adaptation, but in heart failure this becomes disturbed; the elastic arteries with the arterioles and strong left ventricle pass the blood out into the veins more easily than the right heart takes it out of the veins; thus the venous side is congested, and, the bulk of blood remaining the same, the arteries are less full than in health. For the venous system, lying betwixt the systemic capillaries and the pulmonic capillaries, has the right heart for its pump, its propelling power, with some auxiliaries it is true; while the arterial system, extending from the pulmonic capillaries to the systemic capillaries, has for its pump the left ventricle. Failure

in the right ventricle then means an imperfect emptying of the venous reservoirs and venous congestion. This is visible in the colour of the complexion, lips, &c., and the change in colour is of great diagnostic importance. It is more marked in acute attacks of cardiac asthma, or dyspnœa, and passes away to a great extent with the attack. Persistent cyanosis not congenital is ever of bad import, and indicates great debility in the right ventricle. Venous fulness varies with respiration; when the breath is held the veins fill, that is, the arterial system is passing its blood out into the veins faster than the right heart is emptying the veins of blood, the circulation through the lungs being greatly impeded by the arrest in the respiration. A few full breaths, the deep-sighing respiration which follows, soon restore the vascular equilibrium, and the venous fulness departs. Certain venous signs are of special importance, thus jugular pulsation is regarded as pathognomonic of tricuspid regurgitation. In every systole some blood passes backwards ere the auriculo-ventricular valves are closed by the backward current of blood; but as long as the valve is competent this is only to a small extent, but when the tricuspid becomes incompetent, it is a very different matter. The veins then are no longer properly emptied by the failure in their pump, the lungs are congested from defective vis a tergo (asthenic congestion), and the ventricle itself, enlarged and thickened from the obstruction to the blood flow, except in the rare case of primary tricuspid disease, is contracting powerfully but inefficiently from this failure in its valve. The veins, already gorged from this imperfection in their pump, are now subjected to the full force of the ventricular contractions, which are only somewhat modified by the more or less imperfect valve; this is a force which veins were never made to be subjected to, and they speedily yield and become dilated, their valves become incompetent, and each right ventricular systole can be seen in the jugular veins. Considerable venous engorgement and some dilatation even may have preceded the tricuspid failure, but whether or not that failure soon spoils the venous valves and renders them incompetent; jugular pulsation ever indicates that the venous valves are

incompetent, and they are rarely rendered temporarily incompetent, even in very grave attacks of cardiac dyspnœa, consequently experience tells us that when found incompetent, tricuspid regurgitation is present. Tricuspid regurgitation is also, according to all German authorities, accompanied by rythmical pulsation in the liver, from regurgitation admitting of the impulse derived from the right ventricular systole being communicated down the vena cava inferior into the liver, whose veins are then engorged. This rückwirkung (back-working) from tricuspid insufficiency has been especially studied by them, and I have been unable to find any corroboration of Mr. Wilkinson King's theory in their literature.*

Professor Laycock, of Edinburgh, has drawn attention to to the precordial veins in chronic heart disease, and, in some cases, they are certainly dilated, but in many, indeed most cases, there is no precordial venous injection.

Veins often partake of general atheroma, and are full, round, not collapsing, and, in a manner, rigid. This is not only seen in the ordinary subcutaneous veins, but also in the coronary veins, which are often full, tortuous, and somewhat rigid, when the foramina Thebesii have been compressed by distension of the right auricle.

All reference to the cardiograph and smygmograph is purposely omitted, for two reasons—1st. Such reference could only be confusing to the great body of practitioners, who cannot and do not use these instruments; and, 2nd. That they cannot be regarded so much of diagnostic use generally, but, as Traube says,† rather to help us to "new facts;" and there exists no more competent authority, whose opinion is based on knowledge of the instruments and not on a confident ignorance. In addition to these two reasons, some doubt may be fairly entertained how far we yet know

* An American friend is highly sarcastic about "the safety-valve action of the tricuspid," saying that the imperfectness of a valve is the most extraordinary claim on our admiration that he ever heard put forward. It is opposed to the very purpose which valves serve.

† This was in answer to direct question put to him by the writer, when watching him take tracings with his improved cardiograph, in the wards of la Charite, Berlin.

enough of either instrument to warrant the introduction of the subject into a general treatise. No doubt tracings are very indicative to those who understand them, and in time, when, among other things, improvements have been made in the instruments themselves, including some less fallacious plan of applying the knob than by a spring which can be screwed down with a varying force, we may hope to find in these tracings an accurate delineation of the heart's impulse, or of the arterial pulse. But it is to be feared that the tactus eruditus, the trained finger, will then be held in undeserved disregard, for this advance in diagnosis by instruments is far from an unalloyed good.

There is great room for fear that all that power to take in a broad view of a case, with all its points included and fairly appraised, which was so conspicuous in the old school of physicians, and on which they could often base such very successful treatment, is being lost sight of too much now; and that an almost superstitious reverence for instruments of precision is taking its place, leading to undue importance attached to some points, which can be precisely ascertained, and to a certain neglect of other points not to be so closely ascertained, but of equal or even often of greater importance; and consequently a less equal general estimate of the patient's condition and requirements, and of the means whereby the condition may be relieved. This is conspicuous in Germany, where, in spite of a thermometer to indicate temperature, and ice as a direct means of abstracting heat, it is leading to a helplessness and feebleness in therapeutics which, as seen very markedly in the Vienna school, is converting the physician from a healer of the sick, from being of any avail to a patient, the great end of physicians I presume, into a mere scientific observer of morbid action. This is practically having a pathologist, and not a physician, at the bedside.

In addition to the usual means of examining the heart, Flint proposes to add "vocal resonance." It may be most proper to give his own words. "The boundaries of the heart may often be as accurately defined by auscultating the voice as by percussion, and, in conjunction with the latter

method, the former may be resorted to in determining the augmented space which the heart occupies in cases of enlargement. In females often, owing to the size of the mammæ, the diminution or extinction of vocal resonance is more available in determining the area of the superficial region than dulness on percussion."—("Diseases of the Heart," p. 68, 2nd ed., 1870.) There is a practical shrewdness in this which at once recommends itself to the reader.

CHAPTER III.*

Objective Symptoms—Palpitation—Irregularity—Intermittency — Causes — Diagnostic Value — Prognostic Value.

In perfect health the heart's function is performed without consciousness and without evidence of its existence, except after violent effort. Consequently when we find objective symptoms of its existence and its functional labours, we do not regard them as evidences of health, but as the witnesses of disorder and disease. The first of these objective symptoms is palpitation, a symptom common enough both in diseased and in healthy hearts. Palpitation may indicate disorder; it may point to disease. Consequently much confusion has existed, and even yet exists, as to its interpretation. When hypertrophy of the heart was regarded as a disease *per se*, and as a spontaneous increase in bulk on the heart's part, till it became dangerous from its strength, we cannot be surprised that palpitation was regarded as over-action of the heart. How such over-action is possible in a hollow muscle without complete closure and obliteration of the cavities, terminating in death, if at all sustained, it is difficult to imagine. As, however, hypertrophy is found to be nearly absolutely in every case, and possibly yet may be found in every case, to be a compensating growth to enable the heart to fulfil its functions efficiently, so is palpitation found to be an evidence of laboriousness.

Palpitation is quite unlike the steady blow of pure hypertrophy, rythmical and creating no consciousness; it is an unpleasant sensation, the sensation truly of heart taxation. It is brought out readily by a little exertion in the dilated heart, a final argument against its being over-action, for that

* This Chapter is founded on a series of papers contributed by the writer to the *Lancet*.

would be just the time when over-action of the heart would be most perfectly neutralized by the effort. A slight effort in a dilated heart produces what much more violent exertion only evokes in a healthy heart. Can it then be regarded as evidence of weakness or of power? It must be one or the other! Hope long ago pointed out that palpitation in its maximum intensity could be found in a dilated right ventricle at the very time when a patient was dying of cardiac asthma, along with every other indication of failure of power. Since then further observation has corroborated its connection with inability. "This sensation is rather the result of the laborious contractions of an unhypertrophied organ," says Niemeyer. And the same authority states it is found in dilatation before hypertrophy is added, and returns when that hypertrophy is undergoing degeneration. It is the associate of the want of power, not the concomitant of excess of it. That it is found even in hypertrophy cannot be questioned, but it demonstrates that that hypertrophy is insufficient, and is most effectually allayed by agents like digitalis and belladonna, which incite the heart into more perfect contraction instead of paralysing it. It is found along with other evidences of cardiac failure, and the pulse of palpitation bears no resemblance to the incompressible pulse of true hypertrophy. The apparently violent efforts of the heart have no effect upon the radial pulse as part of the arterial system, a sufficient test of the presence or absence of ventricular power. But if the evidence against its being over-action in the diseased heart is complete, how, the reader may fairly ask, do we find it in a perfectly healthy heart?

This perfectly legitimate question can be answered. The centre of the circulation is muscular, and so is the peripheral distribution of the arterial system. This last may be acted upon and so affect the heart. Cyon. von Bezold, Ludwig, and Rutherford have found that the heart beats may be increased when every nerve communication of the heart itself has been severed. By irritating the medulla when the accelerator nerve was unsevered spasm of the arterioles was called out, and with it excited action of the heart.

What, then, do we find, in fact, in the nervous palpitation of the sound heart?

Take a case of hysteria. We find violent palpitation, a small cordy pulse, cold extremities, and, on the cessation of the attack, a profuse flow of straw-coloured urine. We have, then, disturbance of the vaso motor nerves, cold extremities from arteriole spasm, profuse diuresis, the result of increased arterial tension, and palpitation, the evidence of cardiac taxation, in the face of the opposition offered by the spasmodic closure of the arterioles. So in Bright's disease, where Prof. Geo. Johnson has shown that spasm of the arterioles is the exciting cause of hypertrophy of the left ventricle, and that that arteriole spasm is caused by the presence of urine excreta in the blood, so on an exacerbation in Bright's disease, we have those cold extremities, palpitation, profuse diuresis of pale urine in an exactly similar manner. (See Chapter XIII.)

Palpitation, then, even in the sound heart is evidence of taxation of the heart's powers, and this taken along with its presence in cardiac dilatation, and its ready production then by slight effort, will justfy our regarding it as evidence of lack of power, and as such it will be regarded as we go along; and whenever palpitation is mentioned, such is its interpretation.

Palpitation is the first of the three witnesses of cardiac inability, and as a temporary condition, is often found associated along with the more serious manifestations of cardiac failure. Next comes irregularity, and lastly, intermittency. By irregularity is meant here irregularity in time, in rhythm, not irregularity in volume, that is, pathognomonic of mitral regurgitation; though some irregularity in volume is apparent in the more or less imperfect contractions of rythmical irregularity. Irregularity is ventricular hesitation, a halting of the ventricular contraction, which can be explained by a small digression. The sensation of distension by the cardiac ganglia provokes contraction, under the control of the inhibitory fibres of the vagus. This control exercised by the fibres of the vagus secures uniform contraction, and irritation of the vagus can arrest for lengthened periods the

ventricular systole. But while there is a balance of power normally betwixt the sensation of distension and the excitability of the cardiac ganglia, controlled by the vagus fibres, which may be disturbed, and thus irregularity result; there is also a balance of power between the blood to be driven, and the power to drive it. As a matter of fact, irregularity is most commonly associated with this last; is certainly so associated, we may fairly assume, when the other evidences of cardiac inability are present. It may be associated with exhaustion of the sympathetic system of nerves and diminished excitability of the cardiac ganglia. It is an arrest of the ventricular systole, an arrest in the contraction of the fibres, a change of rythm. A prolonged impression on the cardiac ganglia is necessary to excite contraction in the face of the vis inertiæ of the blood column to be overcome, and the restraining action of the vagus. When that contraction is excited, it is short, sharp, and distinct. It is as if the contraction, instead of proceeding from one set of fibres to another, from 1 to 2, 2 to 3, and so on from 6 to 7, the normal number of layers of fibres in the heart wall, had halted during the time of the contraction of the fibres first in action, and then a more than ordinarily synchronous contraction had followed. Irregularity is here used as arrest during a portion of the systole; more prolonged arrest, as during a whole systole, or more, is intermittency. Irregularity then is arrested systole, and when occurring, as is mostly the case, along with other evidences of cardiac failure, and more especially when it is elicited easily by exertion, is an evidence of muscular inability of a serious character, and is a more grave admission of debility than palpitation. When auscultating an irregular heart, we find at shorter or longer intervals a pause. This B. W. Richardson thus describes: "It is like a smith who, striking at the forge a number of strokes in rythmical succession until tired, changes the action for a moment to give a more deliberate and determinate blow, and then rings on again in regular time." The heart beats away pretty rythmically, and suddenly comes this pause, with an indescribable sort of roll over, and on again. The sensation to the auscultator is peculiar and un-

mistakeable, and especially if auscultating without a stethoscope. It has always struck the writer as most resembling the "change" of a horse's feet when cantering; a momentary pause, during which the other foot is put first, and then on again. Whether the contraction of the ventricle at this pause commences from a new set of fibres or not, we will probably never know, but from the analogy to the relief afforded to the smith by the change, and probably so to the horse, we may deem it probable at least. The ventricle certainly halts, and this more too when the patient is exerting himself or in excitement, than when at rest. When Senior Resident Medical Officer of Leeds Dispensary, the writer frequently noticed an irregularity in the pulse if felt when first the patient entered the room, which was either not at all, or in a much less degree, perceptible after a short conversation. When any doubt may exist as to the causation of the irregularity, as to whether it is a disturbance of balance betwixt the vagus and the cardiac ganglia, or a disturbance of balance betwixt the opposition offered in the blood to be driven, and the muscular power to drive it, the question admits of a ready solution. Let the patient make an effort: over the disturbance of nerve balance this has no action; but in increasing the necessity for effort in the heart walls, it has a very perceptible effect in increasing the amount of irregularity; when that irregularity is a disturbance of balance betwixt the blood to be driven and the muscular power to drive it. Frequently this pause is preceded by three or four rapid feeble contractions, then the pause, and rythmical contractions for a time, then the rapid strokes and the pause. This condition is never found but in serious dilatation, and is an indication of a very grave character; it is often well communicated to the radical artery, and more so if the arteries are themselves atheromatous; in this permanent condition, palpitation is readily induced by slight effort.

Last and most serious admission of failure is intermittency, *i.e.*, arrest of ventricular contraction during one or more systolic periods. This prolonged halt in the heart's action has been the subject of much investigation. Since the days

when the brothers Weber first excited it by irritation of the vagus down to the most recent investigations of Rutherford and Richardson, it has been the subject of attentive observation. As to its immediate causation, it has long been regarded as arrest of the ventricle until a second auricular contraction produced sufficient distension to provoke contraction. Of the truth of this, the writer got proof in opening the chest walls of two rats. This was not very skilfully done, and the pulmonary collapse soon finished the rats; but as the rats died and the ventricle intermitted, it could be distinctly seen that the ventricle waited till a second auricular contraction excited ventricular contraction. Moreover, shortly it became manifest that even the second auricular contraction was insufficient to excite contraction, and the ventricle halted until a third auricular contraction excited ventricular activity, and then the auricular contraction passed swiftly on into ventricular contraction. That intermittency of the heart is arrest of the ventricular contraction over one or two auricular contractions may be fairly asserted. It may further be presumed that the increased distension of the ventricular chambers by the extra amount of blood in them from these several auricular contractions, was necessary to excite contraction in the ventricle. In practice then, when we find distinct intermittency, it signifies prolonged ventricular arrest from ventricular inability. In auscultating a heart with fatty degeneration, who, with much experience, has not waited for that lagging ventricle in more than one instance, until a fear has arisen that its arrest is going to be final?

And when found along with other evidences of heart failure, it is a sign of the most serious character: that long ventricular halt is of grave import indeed, and when found along with a diminished first sound, tortuous arteries, panting respiration, and an arcus senilis with a cloudy cornea, is significant enough. It is pathognomonic that the hypertrophy that once existed is going, is indeed far gone, never to return.

We may alleviate the condition somewhat, but more is impossible. We find, too, that such a patient is simply un-

equal to the effort which excites palpitation, as in a heart less structurally diseased. Though its irregularity of rythm may be increased by slight effort, that heart is not equal to palpitation: palpitation is an effort beyond it. Intermittency is, however, not always so associated, and the two most marked cases in the writer's experience, were both connected with aortic obstruction. In these cases the intermittency lasted over no less a period of time than four systoles. The sensations of the patients during this period of ventricular arrest were horrible. The terror excited was visible in their countenances, and quite in accord with Romberg's account of the patient's case given below. The arrest of the ventricular systole before the obstruction offered by the narrowed aortic orifice was pronounced, and enlightens us as to the tendency in aortic obstruction to sudden death. If that halting ventricle becomes structurally unsound, its halt will soon be a permanent one.

Intermittency may then be an evidence of structural degeneration; it may also be present with good hypertrophy even before a very decided obstacle to the flow of blood. It may further be a result of irritation of the vagus, of which a good case is related by Romberg ("Diseases of the Nervous System"), which occurred in Vienna. This heart stood still over a period of six normal beats, at intervals, its rythm being good at other times. A diagnosis was made by Heine and Skoda, that it was due to tumour of the vagus, and when that heart finally stood still, Rokitansky found a tumour like a cherry involving the vagus. The patient's sensations during that halt were indescribably horrible, and a sense of impending dissolution was overwhelming. That this is a rare form of causation of intermittency may be at once taken for granted. Arrest of the heart's action through the vagus by a determined effort of will is not unknown; but it is regarded as a hazardous experiment.

Finally, intermittency may be purely nervous; may be a disturbance of nerve balance, and that only. For an able investigation of this subject we are indebted to the philosophic mind of B. W. Richardson, who has paid great attention to this intricate subject. That Dr. Richardson may have had

occasion to modify his views is merely proof of his earnest wish to arrive at correct conclusions. And though I, in common with the majority of the profession, cannot entirely agree with him in his conclusions as to its divorce from all organic diseases of the heart walls, still a debt of gratitude is due to him for the able and persevering manner in which he has demonstrated that this most serious symptom is not only not invariably associated with grave disease, either in the heart or elsewhere, but that it is often a mere nervous abnormality of no pathological import.

It is well that this fact be known and recognised. On mere intermittency of the heart alone, no practitioner is justified in giving an opinion as to the existence of heart disease. The suffering and misery entailed by hasty medical opinions as to the existence of heart disease of a grave character, and its proneness to sudden death, is something fearful to contemplate. I know well a hale north-country yeoman, of unusually fine physique, whose peace of mind, years ago, was ruined by a rash medical opinion, formed most unjustifiably, and so strong was the impression then made, that no amount of assurance of his health can free him from terrible bondage of this idea.

Dr. Richardson has demonstrated how intermittency may depend upon disease within the encephalon, and when so associated regards it as a symptom of the worst omen. He, further, has collected a most instructive and interesting series of cases where persistent intermittency is the result of shock, of anxiety, as in shipwreck, of grief, &c., in fact of a whole series of psychical troubles, and where this intermittency is quite unconnected with physical disease; and, finally, has recorded cases where, after years of intermittency, no morbid change could be detected, on post mortem examination, to account for this symptom. Hence he has located intermittency in the double nervous system of the heart. This valuable work should put us on our guard against the impression that intermittency is necessarily connected with grave disease of the heart, still it is equally the fact that in the large majority of instances it is so connected; and as to

whether it is so associated, or not, in each case must be determined from consideration of the corollaries.

Finally we have irregularity and intermittency, the common pro-dromata of death. We all form our opinion, in many cases, of how long it will probably take for the sands of life to run out, from the character of the pulse. When the pulse becomes irregular, and then intermits, we know the end is not far distant. So in all serious acute disease this condition of pulse is regarded with great anxiety, and not without reason. As regards the nature of the intermittency we find that in many instances, and in all acute specific disease, and disease of the respiratory organs, the intermittency in the radial pulse is significant of the condition of the right ventricle. Enlightened by the lessons of the dead-house, by the fact that commonly enough the left ventricle is fairly contracted, while the right heart is gorged with blood and distended, we come to learn that this condition of pulse is significant of the condition of the right side, and not of the left side of the heart. The right ventricle is struggling away betwixt the difficulties of opposition offered to the flow of blood in front, of over-distension from the gorged veins behind, and with the numbing, paralysing effect of the venous blood within it, highly charged with carbonic acid, while the left ventricle is labouring under no difficulty in the propulsion of its contents. But many of the fibres of the heart are common to both ventricles, and consequently the left ventricle keeps time with the right, and can only transmit the limited amount of blood coming into it from the pulmonary circulation and the right heart. That this is so is undeniable, but another proof of this can be found by auscultation. The halt of the right ventricle is often pronounced on auscultation before the hesitation in the radial pulse is equally marked. How far intermittency and irregularity in the heart, and consequently in the arteries, is connected with right side failure in some cases, and with left side trouble in others, it is difficult to say. The subject needs investigation. In many cases, where the heart affection lies solely in the right ventricle, as in cardiac asthma, the result of distension

of the right ventricle, not uncommon in persons who have been subjected to prolonged trials of their "wind," as divers, gymnasts, runners,* where the right side of the heart is subjected to sustained strain, the irregularity and intermittency are obviously connected with the right heart, and are much more distinct to auscultation than to the finger on the radial pulse.

The question of the connection and causation of these objective symptoms with nervous affections of the heart, as in Grave's disease, &c., will be considered in Chapter XII.

But their general significance and value in diagnosis and prognosis are as given above, and in such sense and meaning will they be used as we consider each form of cardiac failure, and a remembrance of this will spare the reader much confusion, which might otherwise arise, and spare much reiteration, which would otherwise be called for. In the same spirit must the contents of the next chapter be received and remembered, for though it may be possible in one chapter to review the different consequences of arrested circulation on the different organs of the body, and the different subjective symptoms of heart disease which arise therefrom, it would be well nigh impossible to repeat each in every form of lesion, or even to indicate which most commonly arise. But when arrayed together the reader will not only better comprehend each, but will have a better conception of their relative importance, and of their relation to each other. Consequently it may not be altogether absurd to review the general consequences of heart disease before considering the diseases themselves.

* See Dr. Clifford Allbutt "On Distrain of the Heart." 1872.

CHAPTER IV.

THE CONSEQUENCES OF OBSTRUCTED CIRCULATION, OR THE SUBJECTIVE SYMPTOMS OF HEART DISEASE—PULMONARY CIRCULATION—CEREBRAL CIRCULATION—LIVER—SPLEEN—STOMACH AND INTESTINAL CANAL—KIDNEYS—GENITO-URINARY SYSTEM—SEROUS MEMBRANES—ANASARCA—INABILITY TO SLEEP IN THE RECUMBENT POSTURE—SYMPTOM OF CHEYNE—CONCLUSIONS.

THE various pathological conditions arising from obstructed circulation, and the symptoms to which they give rise, may advantageously be regarded together, and when so collected will more lucidly explain the various symptoms, concomitant and intercurrent affections, which arise during the progress of most forms of heart disease, in a greater or less degree. The importance of placing these altogether is recognised mostly by German writers, who lay great stress upon the "rückwirkung," or "back-working." Affections of the aortic valves are not accompanied by these consequences until the mitral valve becomes affected, whether by extension of the endocardial mischief, or by the valve becoming incompetent by atrophy of the musculi papillaries, or enlargement of the cavity of the left ventricle. Thus in the earlier stages of aortic insufficiency the greatest danger is the risk of rupture of the cerebral arteries and the formation of true apoplexy. But when the mitral valve has become implicated, whether secondarily to aortic disease or originally, then ensue a series of changes and symptoms consequent on the arrest in the circulation. The pulmonary vessels become distended, the right ventricle becomes dilated and hypertrophied, the arrest in the circulation sooner or later extends backwards, and in this order we propose to consider them. First, then, comes dilatation of the pulmonary artery and its branches, thickening of their coats, and ultimately a distinctly atheromatous

degeneration of them, and even fatty degeneration of these altered vessels.

In consequence of this increased bulk of blood constantly in the pulmonic circulation, we have very commonly increase of the connective tissue, or hypertrophy of the lungs passing on to atrophy, deposit of pigment in excess, &c. But more surely as time rolls on we have emphysema. The violent efforts at respiration entailed by the increase of bulk of blood in the thorax, and consequently diminished space for the lungs to play in, thus lead to rupture of the air vesicles, favoured by the altered condition of the lungs themselves, especially in the older and more advanced conditions of degeneration. There is congestion of the bronchial mucous membrane, and effusion of serum and mucous flow; this further adds to the difficulties of respiration. Frequently the pressure on the pulmonic circulation becomes so great, especially betwixt a strong right ventricle and a "button-hole" mitral, that we have rupture of the coats of the vessels and hæmoptysis. This rupture may take place into the structure of the lung itself and cause true pulmonary apoplexy, or the Infarctus Laennecii, the formation of black, round clots of blood the size of a bagatelle-ball. In more advanced conditions we have pluritic effusion. These pathological conditions have as symptoms—impeded respiration, dyspnœa, easily excited by exertion, cough, increased flow of bronchial mucous, hæmoptysis, and the marked tendency to undergo transient exacerbations of exaggeration of all the symptoms, constituting those terrible attacks of cardiac asthma, which add so seriously to the patient's sufferings.

When the effect is felt backwards from the tricuspid valve the venæ cavæ become distended. The venæ cavæ are protected by valves, and thus regurgitation is arrested normally, but in time the distension of the veins renders these valves incompetent. Long, however, before this valvular incompetence has become apparent objectively, there has been a considerable venous congestion, and in this the cerebral circulation takes part. The brain is in the condition of constant excess of venous blood, while it too frequently suffers from a want of sufficient supply of arterial blood. This leads to

excess of connective tissue in the brain, so-called hypertrophy, passing on to atrophy, accompanied by effusion of serum into the ventricles, chronic hydrocephalus, increase in the amount of cerebro-spinal fluid, &c. This venous stagnation has also been regarded as a factor in the production of arterial apoplexy, from the obstruction offered to the flow of blood. Under these circumstances, then, we are not surprised to find mental manifestations not uncommon in heart disease. Most constantly and generally observed in all text books is the frequency of horrible dreams. These torture the little rest such sufferers often are allowed, and more so after a heavy supper, and often precede the attack of cardiac asthma with which the patient wakens. There are, too, constantly, in a greater or less degree, a sensation of swimming in the head, pain, dizziness, vertigo, singing in the ears, ringing, &c., and sensations of unsteadiness. There are, too, the accompanying conditions of mental irritability, want of fixity of purpose, instability, caprice, petulance, and tendency to erroneous reasoning and calculations. Thus the strongest and clearest headed man may become childish, irresolute, timorous, and easily perplexed, or irritable and capricious. These mental alterations add much to the difficulty of treatment, and have not been sufficiently recognised, and many a little unpleasantness, as well as more serious ruptures betwixt patient and medical man might be avoided, if a better general knowledge on the subject existed.

There are often, too, changes in the eye, which cannot be further alluded to here, and variations of vision. While the features are often vascular, congested, with blue lips, &c., and, in the attacks of asthma, even cyanotic, there is often numbness in the hands, and pain down the left arm, and other nervous symptoms, whose proper symptomic value has not yet been appraised, but form a famous field for some energetic worker.

So, too, there are disorders connected with the lower vena cava, and especially the valveless portal circulation. This condition is spoken of generally as abdominal plethora (plethora abdominalis). The various organs thus suffering from venous congestion become altered in structure

and irregular in function. Thus in the liver we find increased connective tissue and cirrhosis; during the progress of this process we naturally find disturbances or liver symptoms, and this, too, in the upper classes, whose means lead naturally enough to the pleasures of the table; not that any positive indulgence may exist, but the weakened organ is less equal to the performance of its function. There is a condition of serous effusion into the bile, termed by Oppolzer "albumicholic," and he further states that in this condition there is congestion of the mucous lining of the bile duct, the formation of jaundice, &c., thereby, and that this, too, is commonly produced by a simple cold. In many instances the liver undergoes venous engorgement, and is much increased in bulk. Altogether the liver is very commonly an organ whose disordered function is one of the most common signals of those changes of structure which follow in the wake of heart disease, and may even constitute the chief symptoms.

The spleen participates in this abdominal plethora, and may almost always be found enlarged; as we do not yet know of any subjective symptoms of spleen affections, no functional manifestions can be described of its engorgement.

But is is very different with the stomach and intestinal canal. Most certainly and surely do we have not only changes in consequence of this venous engorgement, but the manifestations of disturbance have long been known and marked. The symptoms of stomach and bowel derangement in connection with heart disease are to be found in every text-book. The first and most constant affection is catarrh of the stomach, not as an affection *per se*, but rather part of the general venous stagnation and its effect on mucous membranes. This increase in the amount of mucous is a frequent source of trouble. It commonly enough induces indigestion by covering the food taken by a coat of mucus, which renders digestion impossible; so attacks of indigestion are usual and easily provoked, in spite of the care of the patient and the physician. In this condition of the mucous membrane, the disengagement of gas is troublesome. The amount of eructation often seen in elderly subjects with

chronic heart disease is surprising, and the appearance even ludicrous. It is no joke, however, to the patient, and often creates both suffering and alarm, and by pressure on the diaphragm and right heart very unpleasant palpitation is induced. There are, too, in this altered mucous membrane sensations and cravings after highly-spiced and tasty food, often very unsuited to the digestive powers, and these, along with the cerebral symptoms often lead to disagreements of opinion. There is one symptom in connection with this condition of gastric catarrh of great importance, and indeed Dr. King Chambers regards it as pathognomonic of heart disease, and that is the "sensation of fulness" ever experienced. Even though the patient is hungry, and feels so, still that sensation of being already "too full" is there. This is one of the most common concomitant symptoms of heart disease.

The intestinal canal partakes of this general congestion, and furnishes, along with it, disorders. These may be generally divided into two divisions; one, irregularity of the bowels, constipation and its accompanying symptoms from inefficiency in the muscular fibre of the intestinal canal. This is, of course, more common in the large lower bowel and in the rectum, and then indicates enemata and aperients or soap suppositories (Trousseau).

The other division belongs to congestion of the mucous membrane, and its results. Thus, not uncommonly, the patient has passive diarrhœa, often furnishing great relief, and only to be treated when becoming unquestionably excessive. Alternations of constipation and purging frequently occur. This condition of mucous membrane is continued to the formation of piles. Bleeding piles or hæmorrhoids are very common in the sufferers from chronic heart disease, and their bleeding is not uncommonly accompanied by sensations of relief; and not unfrequently when no bleeding has occurred for some time, a flow of blood from another organ may occur, as hæmoptysis for instance.

The effects upon the kidneys are also direct and unmistakeable, but this subject is not so simple, and needs some little consideration. There is a great relationship betwixt

kidney and heart disease, as was observed by Dr. Bright. But while he and most other physicians have regarded the kidney disease as primary, and the heart disease as secondary, some have always maintained that the kidney disease is always and only the result of previously existing heart disease. The connection of heart disease with kidney disease, as its cause, will be fully discussed in a following chapter (Chapter XIII), and here we have merely to do with kidney disease as the consequence of venous congestion due to pre-existing heart disease. Often, indeed, are seen the recent changes in the kidney engrafted on old standing disease. Consequently difficulties have been made about this subject which would long ago have been cleared up, had preconceived opinions not manipulated the evidence. Whether old kidney disease have existed or not, is nothing to the question of the production of recent kidney mischief. When, then, this venous stagnation has led to the usual result, the production of connective tissue in excess, it may be limited to the part of the kidneys remaining sound, or if not altered by previous disease, all the kidney; there is usually albuminuria. When a person without old kidney disease is beginning to suffer from this venous congestion, there are, then, more or less albuminuria, diminished flow of urine, indicating less arterial pressure on the glomerali; the urine is dark-coloured, scanty, and of high specific gravity, with exudation casts or fibrin-cylinders. But where old kidney disease has existed with affection of the left ventricle, a totally opposite series of urine symptoms may have existed. There may have been free flow of pale-coloured urine especially at night, albumen scanty and altogether absent, frequently for lengthened periods; the specific gravity low, and the tube casts granular, small, and contracted. But when sooner or later, if not carried off in the mean time by inter-current disease, the symptoms change, on the failure of compensating power in the heart itself, we have venous stagnation and its consequences, and the symptoms peculiar to that condition. This antagonism betwixt the symptoms of early kidney disease, and the opposite condition when venous stagnation has set in, has led to much confusion; but when the changes are

correctly explained, and for this we must chiefly thank the Germans, there does not exist much real difficulty; the difficulties arose from an imperfect pathology, and more exact observation has removed them. Of course, it is possible to have even old standing kidney disease, the result of old standing heart disease, as we may have old standing secondary disease in other viscera; and further, all kidney complication must necessarily add quickly to the heart's troubles. As the primary affection, so its consequences, when it runs its course quickly the changes in the parenchymatous organs are recent; when its course is chronic so is the character of the changes of structure which follow in its wake. (For further information on the connection of heart and kidney disease, see Chapter XIII.)

Much less discussed, because not so involved, are the diseases of the genito-urinary system. These of course make themselves most apparent in the female sex. This venous stagnation leads to the usual consequences in mucous membranes, and so we have leucorrhœa a prominent symptom. This is natural enough and easy to comprehend, nor is it difficult to understand that those natural periodical discharges of blood from the female genitals are increased in quantity, and even in frequency, by venous congestion of the uterine vessels. Thus menorrhagia is common during the period of a woman's life when she has these discharges, while flows of blood at other times will occur (metorrhagia), and even persist after the cessation of the menstrual flow. Oppolzer was inclined to think that profuse menses often accompanied the arterial fulness of aortic insufficiency, and regarded it as a symptom of some value in that form of valvular disease.

In addition to these, Von Dusch is inclined to regard metritis itself as a result of venous congestion, and it would only be in accordance with the general law of the connection of increase of connective tissue (interstitial inflammation) with venous congestion if this were so. That we must regard the more peculiarly female diseases as the not uncommon consequence of pre-existing heart disease, is inevitable. In the male sex this will lead to vesical catarrh,

prostatic congestion, especially likely to occur in connection with the absence of hæmorrhoids in the distribution of the blood through the hæmorrhoidal vessels; and Oppolzer even goes so far as to attribute hydrocele to congestion of the pampiniform plexus.

This obstruction to the circulation is also felt in the tissues, especially the areolar tissue under the skin, and may show itself at varying intervals, according to the patient's condition. This condition when general is termed anasarca, when limited œdema, but is still popularly termed dropsy (hydrops), and by the laity regarded with great alarm. Nor is this without reason, for this condition is rarely absent from the later stages, and as non-professional people do not investigate processes, but merely mark relationships, its almost invariable presence before death naturally enough inclines them to regard it as an almost certainly fatal omen. It must not, however, be so regarded by us; still its significance is unmistakeable, and it shows that there is an accumulation of water in the blood, probably from the feeble circulation not bringing the blood sufficiently in contact with the excretory organs. It has also been surmised that the retained urine salts in the blood have a tendency to make the fluid portion escape more easily out of the vessels. There is no direct proof for this except that renal dropsy has a more marked tendency to show itself generally, while genuine cardiac dropsy is found in the most dependent parts, and therefore first in the feet and legs. So in general debility and feeble circulation the dropsy, often present, is in the lower extremities.

When there is "puffing under the eyelids," from œdema, there is at once ground for suspecting renal complication with the heart disease. So, too, in the most advanced conditions, when the kidneys almost invariably become affected, there is a tendency in the dropsy to become more general. At first there is a slight œdema over the foot and round the ankle, and a slight "pitting on pressure," *i.e.*, the impression made by the finger persists for some little time, like a pit in dough or putty. This may soon vanish, especially by appropriate means, but sooner or later, may be years under fortu-

nate circumstances, it returns, each time more persistent, and each time more intractable. In more advanced conditions it can only be kept at bay for some limited periods, and soon establishes itself permanently. I purposely use these expressions, as whatever the reader may think when treating a patient with heart disease, the nurses, the friends, and the patient will all form their prognosis from the attitude of the dropsy.

Sometimes, however, dropsy will come on quite suddenly, and when this occurs great relief is often felt, and the other symptoms are naturally relieved by this drain of bulk of fluid away from the gorged veins and right heart. When dropsy does come on quickly, and generally, there is a better prospect of its remaining away for a lengthened period than when it makes its appearance gradually and insidiously. This is an empirical fact, which must not be forgotten in making a prognosis. When the gradually increasing dropsy has been successfully combated for a time by appropriate remedies, and returns again while the patient is under treatment, it is an omen of the worst import. As dropsy proceeds upwards, it involves the knees, thighs, and scrotum; indeed wherever there is areolar tissue and lax skin, it will locate itself in force. Often a temporary improvement in the conditions of the limbs will be found, but along with it evidence of increased distress to the patient. Here there is every reason to suspect temporary œdema of the lungs. An increase in bulk again in the legs will often give relief. The dropsy will then settle itself over the back, shoulders, and in the arms; but by this time there is usually evidence of effusion into all the serous sacs, peritoneum, pleura, pericardium, and cerebral ventricles; carbonic acid poisoning and coma will show themselves, and soon the scene closes. Occasionally œdema glottidis is the immediate cause of death, but not frequently.

During this period of time the skin, stretched by the effusion into the subcutaneous areolar tissue, is liable to certain lesions. On points of pressure it is liable to take on that low form of dermatitis, commonly called "erysipelas," an objectionable expression, as confounding it with genuine erysipelas,

a serious pyrexial disease. So commonly does this inflammation of the skin follow incisions, needle pricks, &c., for the relief of dropsy, that the liability to it forms a strong objection to resort to these measures. The skin, too, is liable to eczematous affections, and sometimes these self-established drains are of great service; at other times they merely add to the patient's sufferings. When there is a combination of heart and kidney disease, eczema is usually present for long periods; but how far the skin is itself affected by this venous congestion at early periods, and what relationship there is betwixt skin affections and obstructed circulation, is not yet worked out. Hebra states that ulcers are ordinarily connected with obstructed circulation, and I have seen very obstinate ulceration in the legs of females with heart affections.

The serous membranes are not usually affected by obstructed circulation, except in the more advanced stages of heart disease; when they are established they add much to the sufferings. When fairly established respiration is only possible in a sitting posture, when the fluids fall away from the lungs and heart by their own gravity.

But inability to breathe in the horizontal posture is not only found in the more advanced stages, but may occur at the earlier periods, especially during paroxysms of asthma (cardiac). It seems often connected with pressure in the diaphragm from the abdominal side of it, and thus pressure on the right ventricle which lies on the thoracic face of it. Thus increase in the contents of the stomach or transverse colon tend to press the diaphragm against the right ventricle, and it is in those conditions where the right ventricle is especially involved that we find relief afforded by the sitting posture. The contents of the abdomen then fall away from the thorax by their own weight, and relief more or less complete is afforded. This is not yet regarded as the absolutely correct interpretation; but it is more probable than any of the older theories, and certainly must, at least, have something to do with it.

Finally, in the last stages of death from heart disease with dropsy, we find the patient becomes subject to carbonic

acid poisoning. The evidence of the accumulation of carbonic acid in the imperfectly depurated blood is drowsiness, increasing gradually, though the patient can be roused to answer questions by more or less importunity, but the eyes soon close, and the patient dozes, with fading respiration, till the necessity for breathing wakes the patient up with looks and sensations of alarm, for that summons to waken and breathe takes the form of a hideous dream. The drowsy patient feels the sensation to sleep so strongly, that even the dread of those horrid dreams is unable to make him resist, and in terror of the consequences he drops off once more into his fitful sleep, again to waken in terror and alarm. There is only one consolation for all this condition, and that is, it is impossible for the human system to sustain it long: that intolerable infliction cannot long be borne.

In connection with this subject is the symptom of Cheyne, quoted by Stokes. The respiration comes and goes in ebbs and flows, gradually deepening into full respiration, then fading away into one or two complete remissions of respiration. It would seem that the exciting cause of respiration could accumulate till a few full respirations can afford complete relief, then the respiration becomes shallower, till it is apparently lost; then the necessity for respiration excites deeper breathing, and so this goes on in a rythmical ebb and flow. Frequently about 15 respirations elapse betwixt minimum ebb and ebb, and betwixt maximum flow and flow. But this, though very common, is not necessarily connected with heart disease, still less with the special form of fatty degeneration; indeed the second good case in my experience occurred in a patient who died of genuine apoplexy, and whose son, in sitting by his father's bedside, having some knowledge of medicine, found his attention largely absorbed by this phenomenon, and made accurate observations on it.

But while the patient suffering from heart disease is liable to all these different affections, it must not be supposed that he must necessarily have them all, or that there may not be periods when he is almost free from all subjective symptoms as long as quiet is maintained; indeed, in some of the milder cases of aortic disease great bodily activity may

be possible without evil result. Niemeyer relates a case where a huntsman in the Griefswald had considerable aortic disease, compensated by hypertrophy, who performed all the marches of the war of 1866 without suffering symptomatically. In some forms of insidious fatty degeneration, indeed, sudden death may carry off the patient before any subjective symptoms have shown themselves, and this, too, especially when death results from effort. But in the great majority of cases more or fewer of these symptoms will show themselves in a more or less marked manner, and certainly before the end, if the patient be not carried off by intercurrent disease. Intercurrent disease is always much more serious in a patient who is already the victim of heart disease, and passing affections are more easily produced by slight causes than in those in perfect health; and, further, these must be more carefully watched and treated.

We may sum up, then, in the four following conclusions:—

1. That when heart disease exists, with venous stagnation, many structural changes in the viscera follow; and that these pathological changes are accompanied by manifestations of functional disorder.

2. Though all functional disturbance, &c., be not the direct result of the heart disease, but have arisen from causes so-called accidental, still these disturbances are more easily produced where chronic heart disease exists than in those who are perfectly sound.

3. That in chronic heart disease the margin betwixt ordinary health and death is lessened; and, consequently, that the limits within which health and ill health may safely oscillate are diminished.

4. Under these circumstances many trivial disorders, which in a healthy person may safely be left to take care of themselves, must be promptly subjected to their appropriate treatment.

CHAPTER V.

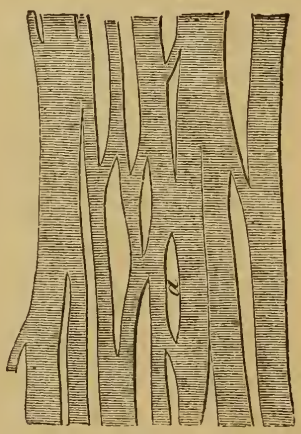
HYPERTROPHY AND DILATATION—HISTOLOGICAL CHANGES—HYPERTROPHY OR HYPERPLASIA—PROBABLE MODE OF GENESIS—CAUSES OR CONDITIONS ALONG WITH WHICH FOUND—OBSTRUCTION—DISTENSION UNDER INCREASE OF PRESSURE—DISPLACEMENT—TEMPORARY CONDITIONS OF FEEBLENESS OF HEART-WALLS—NIEMEYER'S HYPERTROPHY—LIPOMA? TRAUBE'S THREE DIVISIONS; DIAGNOSIS OF EACH—INSPECTION—PALPITATION—PERCUSSION—AUSCULTATION — RIGHT SIDE HYPERTROPHY — SUBJECTIVE SYMPTOMS—PROGNOSIS OF EACH FORM—TREATMENT—QUESTION OF PERMANENCY—IS HYPERTROPHY EVER DESTRUCTIVE?

THIS is perhaps the most important chapter in a work on heart disease, and consequently must be dealt with at some length. It is of the last importance in estimating the prognosis of the great majority of cases. The subject is not altogether free from difficulties, but they will disappear with a little well applied attention, and the chapter is so arranged as to put the different sections in their respective positions to explain themselves, as far as the writer is capable of such arrangement.

The first point is the question of what are the changes in the heart fibre in hypertrophy. These have been stated to be increase in length and breadth, increase in number of fibres, or accumulation of fusiform involuntary fibres, not attaining to the higher condition of striped fibres. This last was largely held in the earlier days of heart pathology. There is a condition somewhat similar to this, but it is that of false hypertrophy. Here there is a development of connective tissue betwixt the muscular bundles of the heart-wall; but, as Sir William Jenner has pointed out, there is no real hypertrophy, no increased power here, but really decrease of it,

by the connective tissue impeding the action of the muscular fibres themselves. In hypertrophy of the heart there is no growth of tissue in any way different from normal heart fibre. Then for a long period it was held that in hypertrophy of the heart the separate fibres were increased in length and breadth—true hypertrophy. This view has been held and taught by Rokitansky and Förster, but they have never been able to demonstrate these changes under the microscope. Then, lastly, it has been stated and demonstrated that the increase in bulk in the heart is due to a true hyperplasia or development of an increased number of fibres in no way differing from the normal fibres. This last view is adopted by Dr. Rindfleisch, and he gives a plate of the muscular fibre in this hyperplasia of which this is a copy.

Fig. IV.



Muscular Fibre of Heart (from Rindfleisch).

The colour of an hypertrophied heart is somewhat darker and redder than a normal heart, firm in its consistency, so long as it is true hypertrophy. The extent to which it may proceed is practically unknown, but it may reach to the size of a bullock's (the cor bovinum), and weigh 40 oz. and more, the ordinary weight of the normal heart being in women 8 oz., and in men 10 oz. We do not know if any corresponding increase has gone on in the nerve supply of the heart. A great deal on this head requires to be cleared up, and in consequence of the difficulties attending the observations, it is not probable that much will be done for some time.

As to the mode in which hypertrophy of the heart is directly induced, it has been held that it was a spontaneous growth to overcome obstruction, always remembering that for some years it was regarded as a disease *per se*, and as such subjected to treatment with the idea of reducing it. But soon it became apparent that in almost every case of hypertrophy of the heart an obvious cause was visible, some obstruction to the flow of blood, as aortic obstruction,

atheroma of the aorta and loss of its elasticity, &c. But, in addition to those, the most marked hypertrophy is found when the heart is exposed to distension under increased pressure, as in aortic regurgitation, where the flaccid left ventricle is distended by a stream of blood driven backwards by the aortic recoil, as well as by the blood coming in from the mitral valve. So too in mitral regurgitation we have dilatation and hypertrophy of the left ventricle when no obstruction is offered to the flow of blood into the aorta. Here we have hypertrophy to resist excessive distension.

We find it, too, when the heart is labouring at a disadvantage as in displacement, or in complete pericardial adhesion. It is also found to follow dilatation from temporary disability of the heart, as after pericarditis and softening of the heart walls by the inflammatory process. In fact, we find it whenever the heart is placed at a disadvantage and is likely to yield to the force of circumstances, and even after yielding hypertrophy steps in and arrests the progress of the dilatation. If we are to read the difficult subject by the light of the recent experiments of Cyon, Ludwig, von Bezold, Brunton, and still more even Rutherford, it would appear that a sensation of distension, of disability, is received by the cardiac ganglia, that that excites a vaso-inhibitory action and dilatation of the vessels of the coronary circulation, and "a broad and more rapid stream" of blood furnishes the material for an increased tissue growth.* This is not certain, but it is highly probable, and is quite in unison with our experience, that hypertrophy more perfectly and quickly occurs in conditions of good general vitality, less perfectly and with more dilatation in less favourable systems, and not at all, remaining simple dilatation, in systems of least resistive power. This explanation of the origin of hypertrophy will be much aided by our reviewing the causes, or, as some may prefer to call them, the conditions which give give rise to it.

Causes.—These may be divided generically into A, Obstruction; B, Displacement; C, Distension under increased pressure; D, Dilatation from temporary causes of debility in the heart-walls themselves.

* See first chapter, section on Cardiac Innervation.

A. Obstruction offered to the flow of blood of whatever nature. When this occurs at all quickly, usually some dilatation takes place before the hypertrophy becomes developed; where it is slow and gradual in its progress, the hypertrophy is often pure and uncombined with any visible dilatation. We may conveniently sub-divide this division into its several parts, which will aid in preventing confusion, which might arise if the different sub-divisions were mixed up together.

1. We have, then, one of the most marked in obstruction offered to the flow of blood from the left ventricle by stenosis of the aortic orifice. This is usually from chronic endarteriitis or atheroma, and is gradual and slow in its growth, and the hypertrophy is usually pure.

2. Aortic aneurism is also a common cause of hypertrophy of the left ventricle, from the obstruction which it offers to the blood stream.

3. Pressure on the aorta by a tumour, or other cause, which presses on the aortic tube and constitutes an obstruction.

4. Aortic atheroma is a common cause of hypertrophy of the left ventricle. When the aorta is thus diseased it loses to a large extent its elasticity, and instead of becoming distended and then by its recoil, due to its own elasticity, promoting the flow of blood, it becomes a comparatively rigid tube, and presents a decided obstacle to the flow of the blood. In other cases the atheroma may be in patches, often annular, and thus by allowing the tube to become distended in some parts and rigidly held of unchanged calibre in others, a decided obstruction is offered. Hypertrophy of the heart sets in to overcome this, and for a time does so often pretty effectually, but it is usually evanescent and wanting in permanancy. As to the connection of this condition with Morbus Brightii, it is not gone into here, because it will naturally belong to the chapter devoted to the consideration of the relation of heart disease and kidney disease to each other.

5. Hypertrophy of the heart is often, almost invariably, found with more or less dilatation, in that curious disease, exophthalmic goitre, Grave's disease, &c., which Trousseau

describes as a neurosis of the sympathetic. Here we find that there is reason to suppose the existence of varying conditions of calibre in the smaller vessels, in some parts contraction, and in others again dilatation, where there is also an obstacle offered to the flow of blood through these tubes of varying calibre. This last cause of impeded flow of blood is not perhaps so certainly settled pathologically as the preceding divisions, which do not admit of doubt. The following division is of great interest, but has only recently attracted its due share of attention.

6. Excessive labour with the arms chiefly. This subject has been more studied in relation to the causation of aortic regurgitation than with its effect upon the heart-walls themselves. More recently Dr. Myers, in the Alexandra Prize Essay, and Dr. Clifford Allbutt, have drawn attention to prolonged strain on the heart itself. The subject is sufficiently important as well as interesting to warrant our devoting some especial attention to it. The prevalence of heart disease, and that too especially of one form of valvular disease in men who follow particular occupations involving the use of the arms, as in hammer-men (strikers), colliers, and others, has been for some time observed. It is common, sadly common, throughout the great industrial regions of England. In the majority of cases the sufferer is exposed to severe, extreme exertion for a brief period, as in a hammerman during the brief period of time that iron remains sufficiently hot to be worked; or in colliers in thin seams of coal, where the collier is working in a constrained position, which from its irksomeness cannot long be maintained. Here we find that while the muscles, all acting towards the trunk, tend to force the venous blood into the great venous centres, the muscles often cross the arteries and impede the flow of arterial blood; indeed Wardrop called this the musculo-cardiac function. Not only so, but the fixed position which these labourers occupy in their work, whether in a constrained position or a fixed attitude, to enable blows to be delivered with greater force and precision, by its muscular rigidity opposes the flow of blood into the muscular system generally. This obstruction leads to hypertrophy of the heart, often

combined with some dilatation, and thus the left ventricle constantly throws a large mass of blood under high pressure into the aorta. In time this, added to the opposition offered to the flow of blood in the arteries, produces such aortic distention and recoil as leads to chronic inflammatory changes in the aortic valves and the establishment of aortic regurgitation and its changes. But from experience among this class the writer is perfectly satisfied that there is a condition of hypertrophy, with more or less dilatation in the left ventricle previous to the development of aortic disease.

Indeed, long before the establishment of aortic disease, these patients will often present themselves for treatment for palpitation, &c., the evidence of laborious action. But as the heart hypertrophies to meet this demand these symptoms pass away, and the hypertrophied, and probably dilated ventricle, throws at each systole an abnormally large bulk of blood into the arterial system. This, by producing increased aortic distension, leads to increased recoil and forcible closing of the aortic valves and disease of them. But in other cases of severe labour, especially in young persons, a condition of dilatation is induced, which often leaves them more or less incapacitated or crippled for labour.

7. Obstruction to the flow of blood in the pulmonary artery. This is a cause of hypertrophy in the right ventricle, and may arise from congenital narrowing, from left side disease, from pressure on the pulmonary artery by aortic aneurism in other pressure, as a tumour, &c.

8. Diseases of the respiratory organs. Chronic diseases of the respiratory organs commonly lead to enlargement of the right ventricle from the obstruction they offer to the flow of the pulmonic circulation. These diseases are chiefly bronchitis, emphysema, cirrhosis of the lung, pleuritic effusion, &c. These two causes, 7 and 8, affect the right side of the heart alone and produce those symptoms peculiar to it which will be found described in a later section of this chapter.

B. The heart also hypertrophies when placed under circumstances where it can only labour at a disadvantage, as when displaced, from whatever cause, or bound down by

pericardial adhesions. When so incommoded, the heart cannot so efficiently perform its function as when in perfect freedom, and so hypertrophy is inaugurated. This is an important section of the causes of hypertrophy, because here we have neither obstruction offered to the flow of blood, nor have we the heart filled under any excessive pressure, the two most commonly recognised causes of hypertrophy. We have the heart merely incommoded in its action by displacement or adhesion, and hypertrophy comes in and enables it to perform its function. Here evidently the disadvantage under which the heart labours tends to its incomplete emptying itself, and soon we would have dilatation, and then the compensatory process of hypertrophy again balances matters. The causes of displacement, and consequent hypertrophy, are the following:—

9. Pleuritic Effusion.—This is the most common of all causes of cardiac displacement. It is usually on the left side, and when so occurring, the heart is displaced to the right side, and the apex may in some cases be found beating under the right nipple. In other cases it is on the right side, and then the heart is forced more to the left of its normal position, and is often tilted up at its apex. Under these circumstances the heart may become ultimately fixed by adhesions to the position which, however, it usually only temporarily occupies. At first, after its displacement, it frequently palpitates, and gives other evidences of its ability, but after a short time it usually becomes evidently hypertrophied, and after that performs its functions with more perfect regularity and efficiency. It may also be displaced by growths within the thorax, by cirrhosis of the lung, and by growth in the abdominal side of the diaphragm, especially in the liver.

10. Rachitis.—This cause of changes in the walls of the heart itself was first definitely pointed out by Rokitansky. He observed that in cases where from deformity in the thorax, usually commencing from the spine, the visceræ within it were displaced, the heart was invariably hypertrophied. Whether this is from mere displacement of the organs simply, or whether an obstruction is created to the flow of blood by the heart being at a new angle with the aorta attached to

the altered spine, or whether the aorta following the spinal curve becomes altered and offers obstruction, or the heart is itself encumbered by being placed in a position not normal to it, is of little moment. Whichever cause is in action, or if, indeed, as is probable, all are, the alteration in the shape of the thoracic cavity is the real cause of the changes inaugurated. This connection of the heart with spinal caries is interesting and instructive. For this is not uncommonly the direct cause of death to these unhappy sufferers, and the sufferer from spinal caries which has become itself arrested, not uncommonly dies with heart symptoms, anasarca, &c., the consequence of obstructed and defective circulation, which takes its origin again in the displaced heart and impeded circulation, a secondary and ulterior consequence of that deformity, which itself had ceased to be any longer a source of danger. This condition of the circulation in sufferers from rachitis should never be overlooked, and this knowledge of the pathological consequences of rachitis will often enable the medical attendant to be of service to the sufferer, which could not be furnished by one unacquainted with this effect of rachitis upon the circulation.

11. Pericardial Adhesion.—The compensatory value of hypertrophy is nowhere better seen than in this condition. Though the pericardial adhesion is sometimes most marked along the track of this coronary circulation (Rokitansky), and thus interferes with the coronary blood stream, and so leads to fatty degeneration, in other cases a quite different result is arrived at. The adhesion may be partial, by bands, which are more or less stretched on each systole, or the adhesion may be complete and the heart firmly encapsuled by the adherent pericardium. So encumbered, the heart beats with difficulty, and empties itself under disadvantages. But in no long time hypertrophy of its muscular walls enables it again once more to fulfil its function, and the palpitation, &c., which marked the earlier stages are no more found. Stokes gives a case where, seven years after an attack of pericarditis, the patient died, and the heart was found perfectly encapsuled, and yet no symptom during life had evidenced that anything abnormal had occurred with the pericardium.

This power to add to its growth and to increase its power on the part of the heart, when incommoded by displacement and restrained by adhesion, is one of the most beautiful instances of the power of the system to compensate evils which are irremediable. That this sensation of inability to contract perfectly should be in itself sufficient to procure increased growth, and thus more perfect function, is an evidence of the great reparative power which the organism possesses. That in cases of general debility this reparative or compensatory effort should be imperfect is no more then may fairly be expected, and is in accordance with all experience of morbid processes which run their course less restrained and checked in organisms of feebler resistent or reparative power than in others where this system is more equal to offering opposition to the downward course.

C. But the most marked hypertrophy with which we are acquainted occurs, not when obstruction is offered to the flow of blood forward, nor yet when the heart is displaced or otherwise interfered with by surroundings, but when its chamber is dilated, on diastole, under excessive, or, at least, increased pressure. Under these circumstances, in a weakly person, with little resistive power, the case assumes the type of dilatation rather than hypertrophy, and quickly passes on to its termination. But ordinarily hypertrophy is the marked characteristic, not without dilatation, however; for increase in the distending power, if at all marked, leads inevitably to increase in the size of the ventricular chamber. But this distension evokes hypertrophy quickly, and to a great extent. In fact no such hypertrophy is met with ordinarily as the hypertrophy of aortic regurgitation, the most marked example. The fact that distension produces an impression which induces hyperplasia is well evidenced here, and the more the pressure within the heart at disastole is increased, as the case goes on and the valvular lesion increases, so is the increase in thickness in the muscular walls. But the increased pressure exercises a steady, distending, and dilating force, which the hypertrophy can only limit for a time, and on the structural integrity of the walls becoming impaired, the process of dilatation is quickly resumed. The

two instances in which this form of hypertrophy is found are aortic regurgitation and mitral insufficiency. That the right ventricle is also filled under increasing pressure in tricuspid insufficiency is certain enough, but as this lesion is rarely found without left side disease, it is difficult to separate the changes which may be due to overfilling from the gorged veins behind the imperfect tricuspid, and the effect of the left side changes. But in the other two cases we have well marked and unmistakeable instances of hypertrophy, solely called out to enable the left ventricle to withstand the increased distending power to which it is subjected.

12. Aortic Regurgitation. This is a familiar form of valvular lesion, and is usually accompanied by most marked hypertrophy and dilatation; indeed in this form of disease is found the cor taurinum. Here no obstacle is offered to the flow of blood forward, but also, unfortunately, but little to flow of blood backward into the ventricle on the aortic systole, or recoil. The blood once thrown into the elastic aorta is normally arrested in the backward direction by the semilunar valves. But when once these become imperfect, then a stream of blood, small at first, but soon larger, is driven back into the ventricle under the power of the aortic recoil. This power far exceeds the force with which the blood comes into the ventricle from the pulmonary ~~veins~~ arteries and auricle. In fact this stream has a wedgelike distending power before which the ventricular walls yield. But hypertrophy is soon inaugurated, and limits the distension by offering an increased mass of muscular fibre to the distending force. This for some time may balance matters, but the imperfect arrest of the blood in its backward course on the aortic recoil in consequence of this aortic insufficiency, leads to imperfect filling of the coronary vessels. Thus, however, the nerve apparatus may try to secure to the muscular walls a more perfect supply of blood, this is prevented, and soon the hypertrophy undergoes consecutive degeneration, and the destructive distension, arrested by the hypertrophy, recommences. Here we see well how the hypertrophy acts in limiting the distension, and also how, when it is arrested, the dilatating process is again inaugurated. For this form

a hundred he is frequently written by Author. The pulmonary veins of two into the left auricle through the mitral valve into the left ventricle. —

of hypertrophy is induced by the distension caused by the addition of the distending force of the blood coming backward from the aorta to the flow of blood coming in normally from the pulmonary veins. But it is under increased pressure of the distending force that we invariably get dilatation along with the hypertrophy. Thus in aortic obstruction the dilatation, if it exists, is so slight as to be unnoticeable, for the increase in the muscular power enables the ventricle to empty itself completely, while in aortic regurgitation the dilatation from the increased distending force is well marked and unmistakeable.

13. Mitral regurgitation. Here, too, we see an instance of hypertrophy, not to enable the ventricle to overcome any resistance to the flow of blood forward, for none exists, but merely to enable it to resist the increased distending force. Here at each systole so much blood escapes backwards through the mitral orifice, this is added to the bulk of blood coming into the left heart from the pulmonary vessels, driven in under an hypertrophied right ventricle behind. This addition causes distension of the vessels behind, and on diastole the blood is poured into the flaccid ventricle under increased pressure. This inevitably leads to distension and dilatation, limited by hypertrophy. In many cases the hypertrophy is early induced, and the cavity of the ventricular chamber is not obviously enlarged, but in other cases, and more commonly, the chamber is increased in bulk, with more or less thickening of the wall. This causation of the changes in the left ventricle in mitral insufficiency, is well pointed out by Niemeyer, and the more the subject is attended to, the more marked does the connection become. This is, too, a very pure instance of hypertrophy coming in to limit dilatation, as no obstacle whatever is offered to the blood stream either at the aortic orifice or in any part of the arterial system. But as the distending force, though increased, is far from having the force of the regurgitant stream in aortic insufficiency, so we see that, though the hypertrophy may be distinct, it never attains those colossal dimensions found, not uncommonly, in the latter form of valvular lesion. This further demonstrates the connection betwixt distension

under increased pressure and the formation of hypertrophy, by establishing the fact of the hypertrophy being proportionate to the distending force; where the force is limited so is the hypertrophy, where the distending force is much increased so is the muscular increase which is called out to arrest it.

D. Hypertrophy following dilatation from temporary causes of debility in the heart walls themselves. Here we have no obstruction offered to the flow of blood, no increased pressure on the flaccid ventricle, no displacement, or other cause of impaired action from surroundings, but compensatory hypertrophy surrounding dilatation, occasioned by temporary causes, and again enabling the heart to fulfil its function comparatively efficiently. The yielding which has taken place in the heart-fibres during a period of debility has, on the health and nutrition being restored, been followed by increased nutrition and hypertrophy. This cause of hypertrophy has not long been recognized, but Niemeyer lays stress on this form of origin of hypertrophy. The chief circumstances under which hypertrophy so rises are various.

14. Myocarditis of a latent character usually accompanies both endo-carditis and pericarditis. But in pericarditis the heart fibres become decidedly softened, for a period remaining some time after the pericarditis itself has vanished. This condition leads to a yielding of the heart walls to the ordinary difficulties offered by the blood-stream, and to the normal distending force of the blood pouring in from the veins, especially in the left heart. Niemeyer has told us how, on the health being restored, hypertrophy gradually surrounds this dilatation, and a compensatory growth endows the heart again with power to fulfil its function.

15. Exhaustion of the sympathetic. This is a common cause of temporary inability in the heart and yielding of its walls. It is obvious that all causes which exhaust the sympathetic, as excessive tobacco smoking, excessive and prolonged debauches, as seen in the feeble, rapid, compressible pulse, common enough in delirium tremens, where the heart is merely pumping a little blood off the top of its contents, but nothing like a good ventricular contraction

occurs; in excessive coitus, in aggravated masturbation, in exhaustion indeed, however produced. Here the heart is feeble, asthenic, irritable, and inefficient, and temporary dilatation ensues.

When this condition of dilatation becomes confirmed on the restoration of the general health by removal of the cause of exhaustion of the sympathetic, hypertrophy sets in. But I am not certain that this new growth may not only enable the heart to contract efficiently, but even lead to reduction of the ventricular cavity to its original and normal dimensions. Two cases tending to corroborate this view came under my notice so as to make an ineffaceable impression. It may not be out of place shortly to relate them.

J. T., an elderly and hardworking woman, was accompanying her husband for a walk in the country one Sunday afternoon. On nearly reaching home he had a violent attack of hœmoptysis, and in twenty days died of acute tuberculosis. She nursed him incessantly day and night; what rest she got being in an arm-chair. We know that there are so many more beats per minute in the sitting than in the reclining posture, and so, of course, so much less rest for the heart. At the end of this time she was much exhausted, and her legs were swollen. A few days afterwards she consulted me, and presented all the physical signs of dilatation, with a rapid, irregular, and feeble pulse. Under treatment, in a few weeks all evidence of heart failure vanished. She resumed her field labour, and eighteen months after this presented no sign of cardiac inability.

T. A., æt. 18, had engaged himself to a farmer for the summer, and overtasked his strength persistently. He came home, quite unable to work, with a weak, irregular pulse, and a dilated heart. After three or four months of treatment (digitalis and iron), he lost all his evil symptoms, and was again a first-class farm servant.

On the other hand, similar cases have remained with obvious dilatation, helped, however, by some hypertrophy, but leaving the patients somewhat incapacitated permanently. This growth of tissue around dilatation arising from temporary causes is a preservative, or rather conserva-

tive action, which we cannot too much admire. That where this causational condition persists the patients should remain with dilated hearts and in a condition of great general debility is no more than we could expect, when the system is unequal to instituting hypertrophy.

These different causes pretty well comprise all the conditions which give rise to hypertrophy. To the list must merely be added two rare causes of hypertrophy. 1. Excessive eating and drinking; and 2. Lipoma (?).

16. Excessive eating and drinking. This cause of hypertrophy is stated by Niemeyer to be found in travelling wine sellers, and Oppolzer states that the increased frequency and activity of the heart's contractions lead in time to distinct hypertrophy. A marked case of this kind occurred recently in the Pathological Institute of the Vienna Krankenhaus, which I fortunately saw. The man came into the hospital with right side paralysis, and soon died with severe hæmorrhage from the mouth and nose. A large clot was found in the left hemisphere, and there was, too, a very large hypertrophied heart of textural soundness, without any apparent cause for it, except his habits. As the arterioles were not examined, it is possible some condition of them existed which might lead to hypertrophy, but the whole subject is obscure.

17. Lipoma (?). Flint relates a case of hypertrophy of the heart, where the heart attained unusual dimensions, in a young man (æt. 23) of temperate habits, who died suddenly with profuse hæmorrhage from the mouth and nose. No cause could be discovered for the hypertrophy in this case, and Dr. Fleischl, one of Prof. Rokitansky's assistants, suggests the possibility that in this case there was a true lipoma of the muscular structure of the heart.

These last two causes are so rare that, practically, they need scarcely be regarded as connected with hypertrophy, and certainly have nothing whatever to do with hypertrophy as associated with dilatation.

All these previously mentioned causes, except the two last, are common to both hypertrophy and dilatation, entirely depending upon the general health, the nutrition, or

the power of resistance as to which of them should predominate. For though certain forms are less associated with dilatation than others, we find that in all, under unfavourable circumstances and where the heart's nutrition is defective, we have dilatation. It is impossible to separate hypertrophy and dilatation in their causation; in fact, it is probable that dilatation, to a greater or less extent, invariably precedes hypertrophy.

While these different causes lead to hypertrophy and dilatation, we have some conditions which scarcely admit of hypertrophy ever occurring, and so may rather be regarded as causes of dilatation merely. These are chronic conditions of debility, and especially those which are connected with imperfect assimilation and persistent exhausting discharges. Under these circumstances it is scarcely possible to expect such a restorative effort as hypertrophy to be possible.

These conditions are chronic dyspepsia, chronic phthisis, chronic uterine disease, chronic diarrhœa, or dysentery, &c., and also we frequently find dilatated hearts in those chronic invalids who seem scarcely capable of maintaining an existence, even under favourable circumstances, and in whom a state approaching to perfect health is simply out of question. What more recent investigations into the action of remedies upon diseased conditions of the heart may be able to do for these unfortunates we can scarcely yet tell, but for many cases there is an element of hope, even if not for others. Often, no doubt, the general condition is dependent on the feeble heart, in others the heart merely partakes of the inherent general feebleness, and in the latter cases there is less room for hope.

But there is one cause of dilatation which must be considered by itself, a most common and most serious cause; and that is degeneration of structure of the heart walls. This occurs sooner or later in the great majority of cases of hypertrophy, and often commences insidiously. It may commence in atheroma of the coronary vessels, or defective nutrition otherwise produced; it may arise, the Germans say, from chronic myocarditis; it is possible that often its origin lies in alterations of the structure of the cardiac

ganglia, but this a mere hypothesis, for of this subject we know positively nothing; but whatever its origin, as soon as it gains a footing the downward changes which the hypertrophy had arrested recommence, we have dilatation and its symptoms, and, as the disease is beyond the reach of any remedy, the case soon passes on from bad to worse to the final change. This is a cause of dilatation for which there is no hypertrophy, no conservative change, and as soon as the degeneration is sufficiently marked to be diagnosed, a prognosis of the worst character is the only one which we can any longer entertain.

We find, then, that from the various causes which induce hypertrophy and dilatation that, according to the nature of the cause, the rapidity with which it is formed, and the general condition of the patient's health and powers, we have different admixtures of the two, in varying proportions. Traube has long classed these as perfect compensation, first form, hypertrophy: imperfect compensation, second form, hypertrophy with dilatation: and no compensation, third form, simple dilatation. This is a practically useful and clinically correct division, though, of course, cases are found of all grades and mixtures.

That pure hypertrophy is almost perfect compensation, so long as it lasts, is certain, but when it begins to yield the case passes from the first to the second division and downwards.

The second division is the most common, and is more or less imperfect, according to the amount of hypertrophy and dilatation comparatively. It is practically sub-divided into the cases which are more correctly hypertrophy with dilatation, and the others dilatation with hypertrophy, according as one or other preponderates. The latter division trenches closely on the last division of simple dilatation. The more hypertrophy the better, invariably, the greater the dilatation the reverse.

I am inclined, however, to think that when dilatation has not long existed that a return to the normal size of the heart is possible; it being impossible to say that there is absolutely no hypertrophy, but with no apparent hypertrophy. In proof of this are the two cases given above, which might

be added to, and also the experience of Fuller, who states that after a three or four years' course of iron he has known all the physical signs and symptoms of dilatation disappear. This return to the normal size has certainly not in any way an *à priori* improbability, for we have every reason to suppose that the heart can become temporarily distended and recover itself, and this is especially well seen in the right ventricle.

Subjective Symptoms.—The subjective symptoms of hypertrophy and dilatation vary considerably. When the hypertrophy is pure the patient is practically as good, that is, as equal to exertion, as an ordinary person, the compensation being perfect. But in the second form of mixed compensation a comparative amount of vigour only is permitted, and the patient feels himself on exertion unequal to the efforts and short of breath, while there is palpitation and not unfrequently irregularity in the pulse. This is of great importance often in forming a diagnosis and prognosis. A heart may present nearly all the appearances of health and yet the diagnosis is doubtful, and some hesitation is felt as to its perfect health. In these cases of doubt let the patient make some muscular efforts, and the doubt exists no longer; there are shortness of breath and irregularity in the pulse, the commencing failure can be detected, and certainty remains. But where the amount of hypertrophy is small, and in the third form, effort becomes simply impossible, and the dyspnœa, and usually cardiac irregularity with palpitation, arrest the patient's efforts at an early period. In fact, a condition of cardiac asthma is soon induced, with lividity of the lips, or even countenance, loss of power, and almost syncope ensues.

The amount of urine passed by patients in these various forms is interesting and instructive. When the hypertrophy is good the amount of urine is normal, when the hypertrophy is mixed with dilatation, the amount of urine is lessened, while in the last form the bulk of urine is scanty and much diminished.

This symptom is one on which non-professional persons lay great stress, and from the amount of urine passed will the patient and his friends calculate the prognosis for them-

selves, especially in more advanced conditions of disease. Nor is this a bad criterion for the practitioner himself, for the bulk of urine depends on the arterial tension, and the arterial tension in turn depends largely on the force of the ventricular contractions.

Objective Symptoms.— The objective symptoms of these conditions are very important, and the correct reading of them is of great use, especially when their import is understood. Thus in perfect hypertrophy we find no objective signs of its existence, and extensive hypertrophy may exist without the patient being conscious of it. But a temporary trial, an increase in the cause, or passing ill-health may lead to palpitation, for which the patient consults his medical man, and the hypertrophy is discovered. But it must always be borne in mind *that in hypertrophy of the heart palpitation is always an evidence that the hypertrophy is insufficient, not that it is excessive.* This is a rule which may be excused being placed in italics, and the remembrance of it will be useful often. The immediate cause of that hypertrophy may be temporary, but the palpitation shows that the heart is also suffering under temporary incompetence. Palpitation is the outward visible sign of internal incompetence, and as such must be so regarded, whether found with hypertrophy or not. The more advanced forms of symptoms of cardiac inability, as irregularity and intermittency, are not commonly found, though prolonged intermittency may occur in aortic obstruction, when the patient is suffering from great debility. Still, the objective symptoms of pure hypertrophy are rather wanting than present, and when present are indications of feebleness. But when there is also dilatation, palpitation is commonly induced by moderate effort, if at all sudden, though all may be perfectly quiet, as long as the patient is at rest, or exercise, or even labour be pursued quietly and steadily without effort. Palpitation, too, is readily produced by gas in the stomach and colon, or by accumulations of more material contents in these viscera. Where the dilatation is the more decided condition, this becomes well marked, and not only is palpitation induced by moderate effort, but irregularity is easily induced, and not unfrequently is found under

all circumstances and persistently. An occasional halt is very common, becoming more frequent on effort, but even a few feeble rapid strokes before the halt are not at all uncommon. And during palpitation itself the irregularity becomes even more marked. The more the objective symptoms of heart failure manifest themselves, the worse the look out of the case. And when the case is one of simple dilatation, all the symptoms are aggravated, and a life of comparative rest is alone tolerable. Here we get not only palpitation and irregularity, the latter ever present, but even intermittency, the long ominous halt on any exertion. The simply dilated heart is ever irregular and unrythmical, with frequent halts and beats of varying duration; often a number of comparatively steady, still feeble beats, then a small cluster of very imperfect contractions, then a distinct pause, and after that the comparatively normal beats are resumed. Sometimes the halt is not single, but repeated even more than once, before the heart resumes its quasi normal beats.

This gradual downward progression is often seen in elderly people when the heart begins to fail, and the first irregular movements on exertion are followed by persistent irregular action, and a permanent halting: then on exertion more aggravated irregularity and the prolonged halt, then the prolonged halt even when at rest, and along with this gradual development of more serious objective symptoms, a steady, downward progress: indeed they go hand in hand together, one illustrating the other.

It may not be possible for the student to recognise these gradations at first, but a little care and comparison of cases will soon give him possession of the alphabet, after which he will soon be able to read complicated cases for himself. But he must always bear in mind that these objective symptoms indicate debility, a fact most difficult to entertain about palpitation, where there is at first sight apparent violent action.

Niemeyer has well pointed out that, in cases of dilatation of the heart from passing causes, the palpitation which accompanied this condition passed away when it was surrounded by hypertrophy, but that when that hypertrophy

was beginning to undergo degeneration, then palpitation showed itself again. And when these objective symptoms do show themselves after hypertrophy has existed some time, their appearance is most ominous. Here, however, the evidences of inability are more apt to show themselves in the more insidious manner of irregularity and intermittency. Palpitation is an active symptom of debility, not commonly associated with degeneration of structure, but rather with the struggles of an incompetent heart of textural soundness. The presence of degeneration is not usually heralded by palpitation, though palpitation is not incompatible with the textural integrity of some fibres, while others are degenerate. Degeneration is rather associated with positive loss of power and instinctive consciousness of failing power in the patient. Degenerate fibres are rather apt to manifest themselves by more or less complete syncope on exertion, or by entire failure of heart action permanently in diastole.

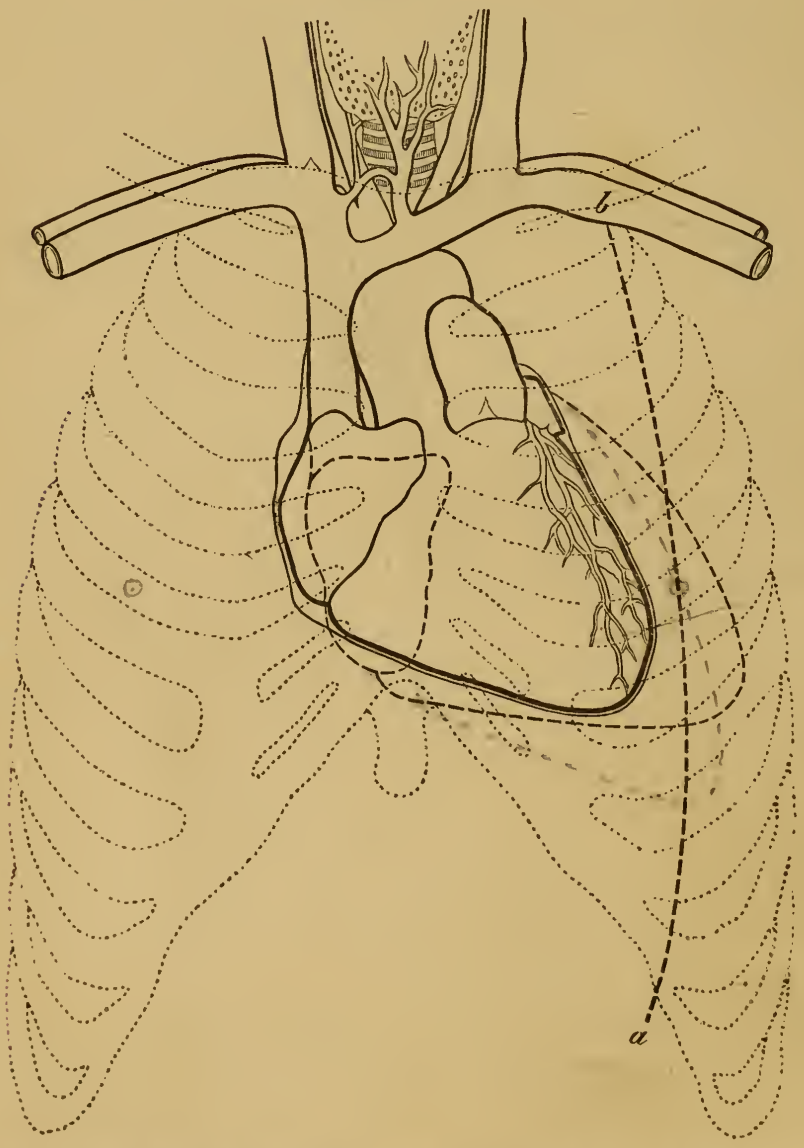
To sum up, we may say that in hypertrophy we have few objective symptoms, rarely amounting to more than palpitation; that in hypertrophy and dilatation we are more apt to have irregularity with palpitation on effort; in dilatation alone irregularity with intermittency increased by effort along with palpitation is more commonly found.

Physical Signs of Hypertrophy.—Inspection.—On inspection an hypertrophied heart will be found with a distinct and localised apex beat considerably lower than the normal apex beat, extending even to the eighth intercostal space, or further, and decidedly to the left of the normal apex beat. This is due to the elongation of the left ventricle, which sometimes extends from one-half to three-quarters of an inch beyond the right ventricle.

This elongation is peculiar to pure hypertrophy, and is not so marked when this hypertrophy is combined with dilatation. On palpation, too, this apex beat is felt well defined, powerful, and distinct. It is felt as a limited point forcibly driven against the thoracic parietes at each ventricular systole. It is quite different from the rounded obtuse heaving formed when hypertrophy is combined with dilatation, and is characteristic of hypertrophy of the left ventricle.

Percussion.—The information afforded by inspection and palpation is corroborated by percussion. There is not such marked increase in lateral dulness as in the direction of the apex of the heart, and to the left. The increase in size and shape in a well-marked hypertrophy, is shown in the accompanying plate, which is copied from von Dusch. The apex is tilted, however, a little too much to the left side.

Fig. V.



Hypertrophy of the left ventricle. The continuous line marks the normal heart; the dotted line the increase. *a*, *b*, Linea mamillaris sinistra.
(From von Dusch.)

Auscultation.—This last gives us very decided information in corroboration of the other means and the information afforded by them. The decided heave, expressive of power,

is communicated distinctly through the stethoscope, or even to the naked ear.

The first sound is not quite so clear, perhaps, as the normal sound, but is loud, somewhat prolonged, and partakes of the character of a "thud." The muscular sound usually covers the sound of the auriculo-ventricular valves, though it is certain enough that their sound must also be increased by the increased force by which they are closed by the thickened ventricle. The aortic second sound, too, is distinctly accentuated when the hypertrophy is associated with obstruction to the blood stream. The increase in power, and the strong muscular tone of the first sound, are of great importance in distinguishing genuine hypertrophy from counterfeits of it. Where associated with valvular lesion, the first sound may be masked, and the diagnosis is formed from the other factors. In aortic disease, the second sound is interfered with, or its place totally supplied by a murmur in aortic regurgitation.

Great aid, too, is derived from the pulse in hypertrophy. It is usually firm, regular, well sustained, and incompressible; indeed it heaves up the finger on each pulsation, much as does the ventricle the stethoscope placed over it.

As mentioned above, in pure hypertrophy there are rarely any of the objective or subjective symptoms; these show themselves when the hypertrophy is failing, or in some instances before the hypertrophy has become developed sufficiently.

Physical Signs of Hypertrophy and Dilatation.—To inspection is visible a more diffused heaving than in true hypertrophy, and this, too, not so markedly towards the left apex. It may extend to the seventh intercostal space, and extend considerably laterally. It is usually distinctly visible in an ordinary person, and very distinct in a thin person. It is, of course, more distinct in the intercostal spaces.

Palpation corroborates the information received from inspection, and the diffused heave is distinct enough to the hand, but it wants the marked force of true hypertrophy. It is more decided, however, than the still more diffused slap of simple dilatation, or where little hypertrophy is present. It is somewhat irregular, and more so after exertion.

Percussion.—By percussion we find that the lateral dulness is decidedly increased, and that instead of the pointed heart of hypertrophy, we have a more obtuse-angled triangle. The dulness extends, too, more to the left of the apex-beat than in hypertrophy, where the left apex is nearly the furthest point to the left.

Auscultation.—In auscultation the first sound is often clear, distinct, more approaching the normal than in hypertrophy alone. The muscular sound is not so preponderating, and the sound of the auriculo-ventricular valves is not unfrequently distinct, and can be clearly distinguished from the muscular sound. The aortic second sound is often clear and distinct, but wants the accentuation of hypertrophy. The action of the heart is often rythmical when the patient is at rest, but easily disturbed by exertion. Where the dilatation is the more marked component, the action is tumultuous, wanting in rythm, and tending to be irregular, a halt taking place at distinct intervals, becoming more frequent on exertion. The objective symptoms in hypertrophy and dilatation are often marked, and palpitation is common.

The pulse is often full but wanting in firmness and rythm, not so sustained, and more compressible than in hypertrophy.

Physical Signs of Dilatation.—To inspection simple dilatation rarely yields any sign, except in very thin persons. In those can be seen a still more diffused beat against the thoracic parietes; most visible in the intercostal spaces, not usually so much below the sixth intercostal space, but decidedly wanting in force. When seen it is commonly irregular. Palpation yields much the same information as inspection, the impulse is felt diffused, wanting in power, rather partaking of the character of a sharp, feeble slap, and extending over a considerable area.

Percussion notes that there is greatly increased lateral dulness rather than increased dulness in the downward direction. The area of complete dulness is considerably increased usually, and the triangular shape of the heart is now rather globular than otherwise. The area of incomplete dulness is largely increased laterally.

The accompanying chart may aid in elucidating this subject.

Nature of change.	Inspection.	Palpation.	Percussion.	Auscultation.	Objective and subjective symptoms.
Hypertrophy.	Distinct, powerful pulsation towards left apex, and in a downward direction.	Limited, powerful pulsation below left apex and to left of it, extending downwards to seventh or eighth intercostal spaces.	Small lateral increase in dulness, but extending downwards and towards.	Strong, dull, muscular first sound, with accentuated aortic second sound. No loss of rythm.	No irregularity. Palpitation occurring when hypertrophy still insufficient. No loss of power and no subjective symptoms. Pulse strong and incompressible.
Hypertrophy and dilatation.	More diffused heaving, not so distinct; more in a lateral than in a downward direction (except in aortic regurgitation).	More diffused and less powerful heaving over apex of heart. Not extending so far downwards and to the left side.	Increased lateral dulness, and extending slightly below ordinary apex.	First sound clear; not so muscular; the valve sound also heard. Some loss of rythm.	Palpitation easily induced. Often a halt in rythm at distinct intervals, increased in frequency by exertion. Power limited. Subjective symptoms more or less marked. Pulse rather full than strong, and compressible.
Simple dilatation.	In thin subjects only can a widely diffused "slap" against the chest walls be seen; chiefly seen in the intercostal spaces.	Feeble diffused "slap" over apex of heart, extending rather laterally than downwards.	Extended dulness, in a lateral direction chiefly; not extending below normal apex beat.	First sound thin and valvular; marked loss of rythm; irregularity almost always present, with a cluster of rapid beats, frequently before the halt.	Palpitation common, and very easily induced. Irregularity persistent, and more marked after exertion, which often leads to the prolonged halt of intermittency. General condition feeble. Pulse small, feeble, and compressible.
Hypertrophy, and dilatation of right heart.	Distinct pulsation over an extensive surface, not in left apex, but across the ensiform cartilage to the right side.	Distinct superficial heaving felt along the region where pulsation is seen on inspection, often without loss of rythm, at other times with irregularity, not so distinct in radial pulse.	Dulness not in direction of left apex, but extending along the edge of the liver, across the mesial line, and over the costal cartilages of of the fourth, fifth, and sixth right ribs.	First sound strong, and muscular accentuation of the pulmonary second sound. Impulse not felt in proportion in radial pulse, and irregularity; when it exists, not so distinct in the arterial system.	Palpitation common with dyspnœa. Often irregularity. Both increased by exertion. Condition that of more or less feebleness, often combined diseases or affections of the respiratory organs. Pulse not affected ordinarily, and when affected, weak. Subjective symptoms usually not long in showing themselves, especially when secondary to left side affections.

Auscultation.—There is something in a dilated heart which, on auscultation, reveals itself by observation. There is a peculiar sensation, of which it is impossible to give a description in words. Either to the ear or stethoscope there is a sort of diffused tumble against the chest wall quite peculiar. It is accompanied ordinarily by an unique sensation of rolling over, with a pause, which B. W. Richardson compares to the sudden halt of strikers on an anvil, which it is not unlike; it has always, however, to myself seemed to resemble most a horse changing its feet when cantering, making a pause, and then going on with the other foot first. It is no doubt a pause which gives relief to the heart fibres. In addition to these the first sound is short, clear, and consists largely of the auriculo-ventricular sound. The impulse to the stethoscope is feeble, and the want of rythm is distinct. The pause occurs at intervals, and is more frequent and repeated on any exertion. Palpitation is common, and is very easily induced, and during it the halt becomes more pronounced and even more prolonged. While the irregular action often met with, consisting of a cluster of short, feeble beats before the pause, is perfectly well marked. When there is also fatty degeneration the first sound is very weak, and there is marked intermittency.

Auricular Hypertrophy and Dilatation.—Under no circumstances do we find the auricles with hypertrophy without dilatation; the opposite condition in the right ventricle is not, however, unknown, and the auricle has been found to consist of the layers of endo- and peri-cardium only with the muscular fibres scattered and separate, so that the auricular wall was partially transparent. Usually, however, we find that there is dilatation with more or less hypertrophy, the thickening often being considerable. The changes in the auricle are almost always connected with disease of the auriculo-ventricular valves. Thus in mitral disease the left auricle becomes thickened as well as enlarged by the reflux, or insufficiency, and by the opposition offered in mitral stenosis. This hypertrophy in stenosis, no doubt aids to bring out the pre-systolic murmur, which is not, however, always heard. This may be due to the fact that in mitral

stenosis the endo-cardium of the auricle often becomes thickened, opaque, and frequently sufficiently rigid to interfere considerably with the auricular contractions. The hypertrophy of the auricles is almost solely to resist distension, for the large orifice by which the veins empty themselves into the auricles offers a ready channel for regurgitation on the auricular contraction. Still, the fibres of the auricle do contract towards the auriculo-ventricular orifice, and thus the direction of the contraction is favourable to the propelling of the blood towards the ventricle. The condition of the auricles it is not at all easy to determine during life, and it is usually surmised from the surroundings with which it is found associated, as corroborated by what is found in the autopsies. Of the left auricle, indeed, we can ascertain very little positively, as it lies so behind the base of the heart, and with the amount of lung covering it, any attempt to map it out by percussion must almost be necessarily schematic; while as to any other direct way of examining it, we possess absolutely none. So the left auricle can scarcely be said to have any physical signs connected with it. The right auricle is better within reach, and will be considered in the next section.

Physical Signs of Changes in the Right Heart.—Hypertrophy is always, in the right heart, connected and found along with dilatation; and is the common concomitant of left side disease, but not necessarily of left side hypertrophy. It depends on the integrity of the mitral valve almost exclusively as to whether right side changes should accompany left side changes, or not. But the right side of the heart may become hypertrophied without change in the left ventricle, and that too not only in mitral stenosis, but in cases of lung affections and other affections of the respiratory organs, as well as those cases where obstruction is offered to the flow through the pulmonary artery. The right ventricle has a tendency too to be enlarged and thickened in swimmers, divers, gymnasts, runners, &c., who tax the wind considerably. This obstruction to the flow of blood causes changes in the right ventricle, and as long as the right ventricle grows and strengthens the wind becomes longer, or, in swimmers'

phrase, they "train on." But when the efforts become too prolonged and severe, the right ventricle becomes distended and less capable of forcing forward the blood, the wind becomes shorter, or, as the swimmers say, they "train off." From these different causes the right ventricle alone may become dilated and hypertrophied, and it must be remembered that in consequence of its shape and evolution the right ventricle is more prone to dilatation than the left. It has been said, not untruly, that hypertrophy is the characteristic of the left ventricle, and dilatation of the right ventricle. Clinically this is the fact. In this condition of the right ventricle the heart becomes much more relatively altered in its position than in left side hypertrophy. It lies horizontally almost in the chest, while, instead of the apex being the most dependent part, a line may almost be drawn from the apex across the ensiform cartilage to the cartilages of the fifth and sixth right ribs. In fact the heart becomes more an obtuse triangle lying upon its side than anything else.

The position and relations of the heart in this condition is well given in the accompanying plate from von Dusch:—

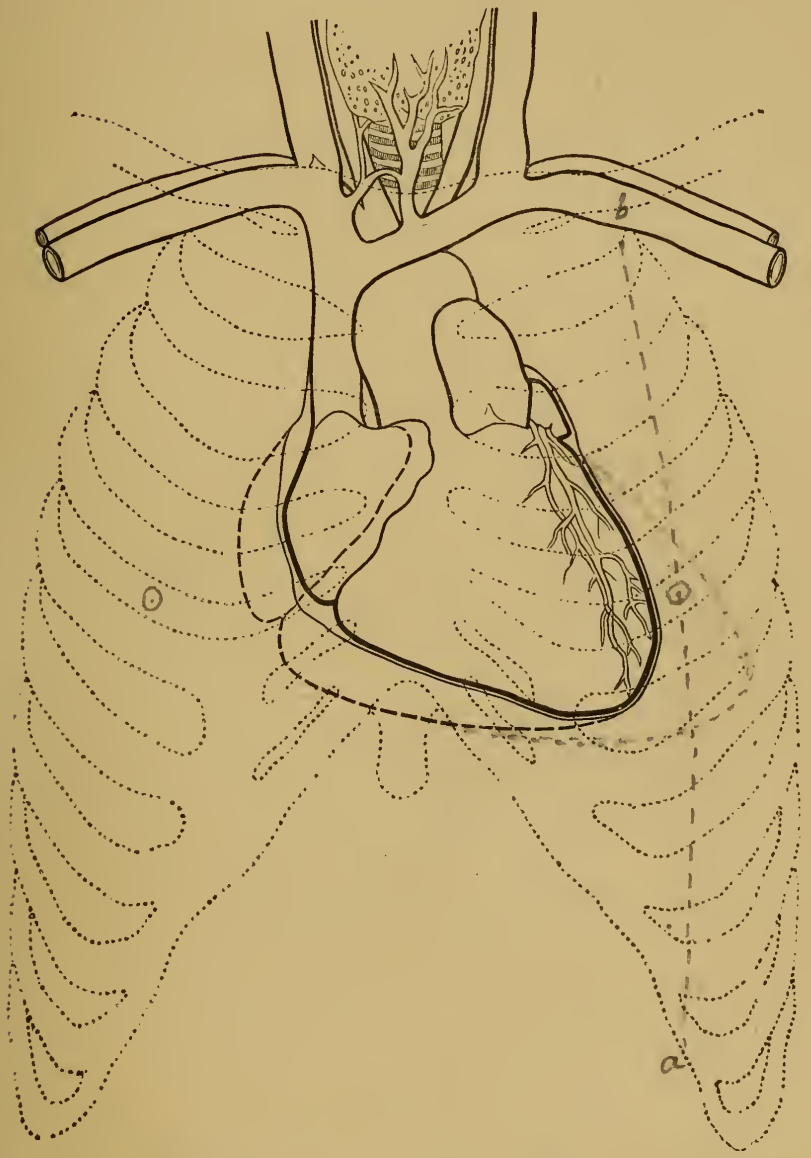
To inspection, the beat of the heart is usually pretty distinct, diffused, and extending from the right apex towards, and even beyond, the ensiform cartilage. There is commonly enough no action seen towards the left apex, and, indeed, in describing the signs here the condition of the left side will be assumed to be normal. When there is both left and right side hypertrophy, the characters of both will be found combined, and somewhat modifying each other.

Palpation notes that there is a diffused heaving, very distinct, superficial, and not without force, extending from the right apex in a direct line towards the third, fourth, and fifth costal cartilages on the right side. It is not uncommonly accompanied by a distinct pulsation towards the second and third right ribs, which is indeed sometimes visible, and which is caused by the action of the enlarged right auricle.

Percussion.—This is a somewhat difficult matter when we come to the right side of the heart. Firstly, however, we get the negative evidence of the absence of left side enlarge-

ment, and the positive evidence that the apex beat is not lower than its usual position, which make it pretty certain

Fig. VI.



Hypertrophy of right ventricle. The dotted outline shows the increase over the normal size, which is marked by the continuous line.

that we have not left side hypertrophy to deal with. Thus the increased action observed by inspection and palpation must be, by exclusion, right side. Then there is the increased dulness extending downwards more extensively than is normal. But it is not safe to be very dogmatic about the distinction betwixt heart and liver dulness along this edge, and the action to palpation and the sound to auscultation are safer guides, at least to the beginner. Still by percus-

sion can be found a dulness, more than normal, extending to the right of the sternum and along the area delineated in the engraving. Very often from the second rib to the third intercostal space can be found an area of dulness corresponding to the enlarged right auricle. The area of increased dulness is distinctly towards the right side of the chest, extending to the right of the normal dulness, and corresponding to the evidence furnished by the other means of examining the heart. The positive evidence furnished by these three means, and the absence of evidence of left side enlargement, constitute a fair ground for a diagnosis.

Auscultation.—On auscultation the first sound is heard distinct and strong, with a sensation of superficiality about it which separates it from the deeper seated sound of the left ventricle. There is heard, too, at the ensiform cartilage, often distinctly, the sound of the flapping together of the valves of the tricuspid. This last is a point of some importance in distinguishing hypertrophy of the right side. There is also heard, too, the accentuation of the second sound at the pulmonary valves. This point is of even more importance in the formation of the diagnosis, and Skoda regarded it as almost pathognomic of increase of size and power of the right ventricle. These are the positive signs which guide us, and there is the absence of evidences of the hypertrophy being in the left side, as the non-effect upon the pulse, for instance. This is a point to be attended to in the establishment of the hypertrophy being upon the right side. There is obviously increased action in the heart, not detectible in the radial pulse, which would be the case if it were seated in the left ventricle. There is not uncommonly excited action or palpitation of this hypertrophied chamber, and even irregularity in it, not traceable in the radial pulse. It is not unusual to find, when the right side is affected, a somewhat distinct irregularity in the heart to the stethoscope, when there is no corresponding irregularity in the pulse, and at other times a much more decided irregularity in the heart than is to be found in the radial pulse, as if the left ventricle only transmitted imperfectly the irregularity of the right side. There is, too, great tendency to cardiac dyspnœa,

induced by slight causes, and not unfrequently with this condition of the right side, chronic affections of the respiratory organs, especially such affections as impede the flow of blood, as emphysema, chronic pleuritis, or rather pleuritic effusion, interstitial pneumonia, &c. The subjective symptoms of heart disease are never far distant, and exist in a modified form even with tricuspid sufficiency, and, when the tricuspid does become affected, follow quickly, with the jugular pulsation, which is pathognomonic of tricuspid insufficiency.

The liver-pulsation which accompanies this condition, and which may be caused either by venous regurgitation into the liver, as stated, or by the systole of the right ventricle being communicated through the diaphragm which separates it from the liver, is a point on which the Germans lay great stress, and needs investigation by others.

These different points by which hypertrophy and dilatation of the right side may be approximately distinguished have been given somewhat at length, but it is to be hoped not unnecessarily so. For the certainty of the right side being so altered is often an element of hope, as regards time, in left-side failures; it is important often as showing the effect of diseases of the respiratory organs; and is finally often a point to be noted as its subsequent failure, or passing away, or the addition of tricuspid failure, are omens of the most unfavourable import.

Prognosis.—This is a somewhat complicated subject, as the various conditions of hypertrophy, hypertrophy with dilatation, and simple dilatation must always be regarded in relation to their cause, to conditions which may have arisen during their course, and their surroundings, the patient's age, general nutrition, family history, &c. In making a prognosis in each individual case all these collaterals must be considered, but in speaking generally of the prognosis of these three conditions we must refer to the condition of the heart itself.

In hypertrophy we find that the conservative effort has a distinct tendency to maintain itself, by comparison with the other less perfect forms of compensation. The more complete compensation too not only is good in a prognostic

view as regards duration of life, but also as regards bodily power. In cases of obstruction to the flow of blood and of displacement, perfect compensation may be attained and maintained for years. Still the person could no longer be considered a good life from an insurance point of view. In other cases the compensation may be of only a temporary character and soon pass away, as by the occurrence of atheroma, by perverted or defective nutrition, or other cause by which the nutrition of the heart may be affected.

In the absence of these causes of failure of the hypertrophy, a fair amount of health and vigour may be maintained for years, and indeed as long as the hypertrophy can be maintained. This is more important for those who must take a considerable amount of active exertion in order to procure a living, than those whose means enable them to take life quietly. Where exertion is unavoidable, directions should be given to reduce it to the minimum, for the hypertrophy will wear out soon enough without hastening the event unnecessarily. Even under these circumstances, the patient may maintain his ground for years, especially with the aid of a little treatment occasionally. But in making a prognosis we may often speak with confidence that no untoward result may be anticipated for a considerable time at least, but repeated examination at intervals is desirable. Hypertrophy is, however, liable to the risk of apoplexy, especially when the arterial tension is increasing, and this possibility must not be forgotten. Altogether, hypertrophy is favourable to a more prolonged existence than is possible under any other circumstances.

Hypertrophy with dilatation is assuredly a less perfect form of compensation, and as such is neither so favourable to duration of life, nor to capacity to undergo exertion. The comparative amount of dilatation and hypertrophy and its mode of origin, must guide us: where hypertrophy has grown around dilatation from temporary causes of cardiac inability, the prognosis is infinitely better than where hypertrophy is yielding, and dilatation setting in.

The amount of exertion which is unavoidable is of great moment here, and all exertion exercises a malign influence

over the progress of the case. The avoidance of exertion affects the prognosis favourably. Here the effect of nutrition too is marked, and any temporary failure of nutrition has an undesirable effect. All causes, indeed, which tend to increase that dilatation which the hypertrophy has arrested, affect the prognosis, and should, if possible, be removed. In itself, hypertrophy with dilatation carries with it a much worse prognosis than hypertrophy, in direct proportion to the amount of dilatation. There is no risk of apoplexy here, but some risk of cardiac failure. Slight exertion is apt to induce unpleasant symptoms, and temporary causes of exhaustion may be accompanied by alarming attacks of syncope.

In very favourable cases some capacity for exertion may exist, but only to a limited extent, and the patient is maimed for life, *lateri hæsit lethalis arundo.* In giving a prognosis, it must be guarded as to time, and the importance of avoiding causes which would tend to aggravate the disease must be laid down with stress. Even with all care, the case is an unfavourable one, though life may be prolonged for years under favourable circumstances, for any intercurrent disease is much more dangerous than to a healthy person. Rokitansky holds an opinion that heart disease is protective against phthisis and other diseases, but we must remember that he is a pure pathologist and not a physician. All acute disease is very dangerous to a person with hypertrophy combined with dilatation, and may soon induce an increase in the dilatation. In elderly persons who commence to feel less equal to exertion, and in whom the heart is discovered dilated even with considerable hypertrophy, the prognosis is serious to life, hopeless as to cure, and if this advance in spite of treatment, a fatal event is not far distant.

Dilatation, simple and without hypertrophy, is a condition much worse, in every sense as well as prognostic, than the two preceding divisions. There is not only incapacity for all exertion almost, but the prognosis as to life is very unfavourable. This feeble heart even during rest gives evidences of its inability, and these are much increased by any necessity for exertion. Fortunately, however, the general condition is such that exertion is rarely possible, and thus the

risks arising from it are avoided. The case is rarely without more or fewer of the subjective symptoms of venous stagnation, and the congestion of the abdominal viscera renders the sufferer an almost constant dependent on therapeutic aid for some visceral complication or another. The condition of dilatation is unfavourable even to its own maintenance, and the oncoming of structural degeneration is ever threatening. For this dilated heart is ever contracting feebly and inefficiently, and only pumping a small quantity off the top of its contents into the aorta at each systole. The aorta is but imperfectly distended, and therefore its recoil is slight; this aortic recoil is the propelling power into the coronary arteries, and the diminished recoil entails an imperfect coronary circulation, and that, in its turn, imperfect nutrition of the heart itself. This is a great source of danger in dilatation, while cardiac syncope on exertion is another: indeed, the danger of this syncope being fatal, depends much on whether the walls are still structurally sound, or not. The effect of intercurrent disease here is even more marked than in the preceding division, and acute bronchitis is especially liable to be fatal. The patient is indeed more or less a perfect invalid, and the prognosis to life is very unfavourable. One class of cases alone have a prognosis not so very unfavourable to life, and that is the elderly lady with means, who often maintains an existence for years with a heart that would be fatal to a seamstress in a few months. The prognosis of simple dilatation is necessarily bad, and if the symptoms of advancing degeneration of the heart fibre be added to it, the duration of life is limited. Often in elderly persons the advance of degeneration of structure will manifest itself in dilatation with unfavourable objective symptoms and increasing debility, and here the prognosis is of the most unfavourable nature. In simple dilatation, palpitation is almost a favourable symptom, as being to some extent an evidence of structural soundness, while persistent irregularity, with intermittency and attacks of syncope on exertion, is of the worst omen.

Can hypertrophy ever be regarded as itself destructive? This is a question of some considerable importance, and must

be considered before any allusion can be made to the treatment of these conditions. When it is to overcome obstruction offered to the flow of blood, when it is to enable the heart to contract more efficiently when hampered by surroundings, when it is to enable a dilated heart to act more perfectly, we may safely say it is not. But hypertrophy, and that too to a very decided extent, occurs under one other set of circumstances, where its presence, though absolutely necessary on one hand, certainly does mischief on another, and that is when it is evoked to resist excessive distension.

This occurs either in mitral insufficiency or in aortic regurgitation. In mitral regurgitation the hypertrophy is rarely excessive, and even when considerable and combined with dilatation, it is rarely destructive to the arterial system: this is probably due to the escape of so much blood backwards through the defective mitral at each systole. But in aortic regurgitation the case is much different, the increased power with which the flaccid ventricle is distended by the additional blood coming in under the force of the aortic recoil, leads to dilatation which is only arrested by massive hypertrophy. This dilatation and hypertrophy is usually excessive, and at each systole a bulk of blood considerably exceeding the normal, and often twice as large, is forced into the arterial system under greatly increased pressure. This leads to excessive, even if instantaneous distension of the arterial system, as is well shown in a smygmographic tracing, or evidenced to a finger placed on an artery. This excessive distension leads to chronic thickening of the endarterium or endarteriitis deformans, and general degeneration of the arterial system. Not unfrequently this distension leads to rupture of a cerebral vessel and thus to death. But this general affection of the arterial system leads to loss of elasticity, and thus to loss of recoil; the coronary circulation is thus diminished, an effect much aided by the imperfect closure of the aortic valves, and the hypertrophied heart soon again becomes the subject of that dilatation which it can no longer arrest in consequence of the degeneration of its structure. Still even here the greatest danger is the loss of hypertrophy, so that no treatment to reduce that hyper-

trophy, even granting that it were possible, could not be justified, as avoiding the Scylla of arterial destruction could only run us on the Charybdis of cardiac failure.

Question of Permanency of Hypertrophy.—This is an important question, and lies midway betwixt prognosis and treatment, for if affected by treatment this must modify the prognosis. The permanency of hypertrophy is of course a question of comparativeness, and depends much on the surroundings. We have just seen that it is of brief duration in aortic regurgitation, and that too for causes which we can readily understand. Under other circumstances it is usually more permanent, next least perhaps when evoked to overcome the obstruction offered by atheroma.

For here again the loss of aortic elasticity impairs the coronary circulation, and leads to structural degeneration. Under other circumstances it is usually more persistent, and may continue unaffected for years, and thus enable the patient to maintain a fair existence. Its maintenance depends much on the general nutrition, and consequently in poor people it is not so permanent as in those who can procure good and sufficient food. It is more permanent in those, too, who can maintain themselves without severe labour, which tends soon to wear out the compensatory growth. Flint expresses a decided preference for a moderate amount of exercise in all persons who are the subjects of heart disease, and states that their course is quickly downward where exercise is no longer taken. I do not think that this statement is liable to the obvious objection that their no longer taking excercise is the result of an increase in their heart affection, a construction which might be put upon it, but rather that a moderate amount of exercise is favourable to the maintenance of the general health and nutrition, on which so much depends. When we consider how intimately associated the heart's labour is with exertion, we must see that such exercise should be of that character as is least likely to tend to fatigue and exhaustion.

Treatment.—Hypertrophy. In speaking of hypertrophy and its treatment, after what has been said above as to its causation and its nature, it is scarcely necessary to add that

all ideas of treatment, based on the assumption that hypertrophy can be reduced by remedial measures, must be abandoned. In the first place it is very highly questionable if it be possible at all, except by the removal of its causation, and for this latter there is some authority, as, according to Larcher and Ducrest, the hypertrophy of pregnancy passes away on parturition. But all plans of reducing hypertrophy by the starvation plan, founded on Albertini and Valsalva's treatment of aneurism, or by iodide of potassium, &c., must be regarded as, fortunately for the patient, inoperative and inert ; for, were it possible, it would merely substitute for hypertrophy the much more serious condition of dilatation which that very hypertrophy holds at bay, but which, so soon as the hypertrophy is impaired, will immediately recommence. Whatever troubles may be immediately caused by hypertrophy, as in aortic regurgitation, must be submitted to, for though rupture of an artery is not uncommon, by far the most frequent cause of death in this condition is failure of the hypertrophy. So to in atheroma of the arteries, as long as there is good hypertrophy there is some risk of apoplexy, but how much is the danger to life increased by the combination of atheroma and dilatation, and the most perfect hypertrophy in this condition all too soon is lost in degeneration of structure from imperfect nutrition; the diminished recoil of those rigid tubes no longer filling the coronary circulation. In hypertrophy the only treatment is such as is likely to maintain it in its integrity, or, when it is not quite perfectly compensating, such treatment as will conduce to more perfect hypertrophy. In these conditions of not quite perfect hypertrophy there is palpitation, easily induced by exertion. Here tonics, especially such as act directly upon the heart, and produce more perfect ventricular contraction, as digitalis, belladonna, &c., are indicated, and in practice give prompt relief to the symptoms. But it must ever be borne in mind that in these conditions, where the system unaided has nearly been able again to strike a balance of powers by hypertrophy, much less of these agents will achieve the desired result than where hypertrophy is combined with dilatation, a decided evidence of inability, and still more where

hypertrophy is wanting altogether. Thus poisonous symptoms from comparatively small doses of digitalis are far from uncommon where it is carelessly prescribed in large doses in hypertrophy, while its use in much larger doses, continued uninterruptedly for a considerable time in dilatation, is not accompanied by any such symptoms of poisoning. To this truly specific treatment may be added iron, vegetable tonics, a liberal diet, and indeed all such measures as are favourable to nutrition.

Hypertrophy with Dilatation.—Here decidedly more active measures for the improvement and strengthening of the heart walls are not only indicated but demanded. Much more digitalis may be administered along with quinine, iron, or strychnine, and not rarely its accompaniment by arsenic, iron, and cod-liver oil is indicated. For here we have imperfect hypertrophy, not of a temporary, but of a permanent character to deal with. Consequently, agents which will directly increase the power and completeness of the ventricular contractions are of the greatest service, and may, and often must, be continued for prolonged periods of time, months indeed, uninterruptedly. Whether a return to the normal size of the dilated ventricles is often attainable is perhaps doubtful, but certainly a much greater proportion of hypertrophy can be so secured, and that is no slight matter. For the more hypertrophy, the more perfect compensation, and the less the liability to the recurrence of dilatation, as well as the more bodily vigour, and the condition of general health. Hypertrophy tends to maintain itself, while the tendency of dilatation is as unquestionably to the inauguration of degeneration. In this combined condition there is less capacity for exertion than in pure hypertrophy, and the heart is more easily affected by exertion, and palpitation is induced from slighter causes. Consequently exercise and labour can only be permitted to a limited extent, and if freely indulged in can only have the effect of hastening the date when that hypertrophy will become imperfect, and the dilatation arrested by its return. Thus it is desirable that these patients should have their crippled condition explained to them, and the necessity for limited exertion insisted on;

the more too because the baneful consequences are not so much immediate, but rather remote, and thus apt to be neglected.

In that form of hypertrophy with dilatation in elderly persons, where are found irregularity at rest and palpitation in slight effort, with increasing failure of the powers generally, which has been before alluded to, all treatment is but temporary in its character, and rather palliative than curative. This is not difficult to understand when we remember the pathology, and consider that here there was in all probability hypertrophy, which had given no sign, and that now this hypertrophy is yielding to structural degeneration, which permits of dilatation coming on, and which that dilatation only aggravates, and the combination of the two quickly bring about a fatal termination, which all and every treatment is equally unable to avert.*

Dilatation.—The treatment of dilatation taxes all the powers that can be brought to bear on it, and the success which attends our efforts is very limited indeed, when regarded from a curative point of view, and not very satisfactory by any means when regarded from a palliative stand-point merely. That more cheering results will follow from recent investigations are within the limits of hope, when we know that we do possess agents which not only conduce to more perfect ventricular contraction, but by whose aid hypertrophy may be furthered, if not indeed directly induced and fostered. In many cases of temporary debility leading to heart failure this is not so problematical, and we possess good grounds for entertaining the view that compensatory and conservative changes may be inaugurated and maintained by remedial agents, and an appropriate diet. But while thus hoping about the future of some cases, we must admit that in the majority of cases no such ground for hope exists, and all that

* A friendly critic, in reviewing my recent essay "On Digitalis: its Mode of Action, and its Use," asks me "to distinguish for him those cases in which digitalis seems at first sight the proper remedy, but in which it fails and disappoints us." This question it is not easy to venture to answer, but I am of opinion that the failure often lies in the fact that the case is one where degeneration of the muscular fibres of the heart, of whatever kind, is underlying the condition presented to us for examination and treatment.

the most sanguine can hope for is that the destructive changes may become arrested and life be prolonged. This prolongation of life is only compatible with the absence of all exertion, and with a life of comparative quiet. Consequently the prognosis amongst the poorer classes is very bad, and our means of relieving them very inadequate to the necessities of the case. Rest, nutritious and easily-digestible diet, are absolutely requisite, and a residence in a hospital ward for a few weeks often enables the sufferers to work on for some time after in comparative comfort. These patients are much benefited by a residence in a convalescent hospital, provided that they have not much to do, or can be almost entirely excused from labour. In patients of comparative means, the effect of rest, absence from all exertion, and appropriate treatment is often very encouraging. There is always, however, less hope in a case where there also exists a valvular lesion. The addition of a valvular lesion subsequently was at one time a source of fear, on the theory of the walls yielding until the auriculo-ventricular valves were no longer sufficient; but Rokitansky states that in cases of dilatation the valves and their attachments also stretch, so that such incompetency does not occur in fact.

As to the treatment to be adopted, in addition to rest and appropriate diet, it is a difficult subject, as the peculiarities of each case must be studied, and the treatment adapted to them. All discharges, that are at all exhausting, must be attended to, and as these are often the consequence of the defective circulation, treatment of the heart itself often is of material service in aiding the effect of the local remedies. The more especial treatment consists of digitalis in combination with vegetable tonics, mineral acids, or chalybeates, according to the indications: when the appetite is feeble, and the stomach not quite what could be wished, the addition of the vegetable tonics and acids is indicated: when digestion is good, iron may be combined with digitalis, and the general nutrition aided. When there is much acidity in the stomach, the iron may be advantageously exchanged for alkalies for a time, or some preparation of it resorted to which goes well with alkalies, as the potassio-tartrate of iron.

Cod liver oil may be tried where it does not disagree with the stomach, and is a well-known useful adjunct in helping the formation of muscular fibre. Where it disagrees, cream or some of the new preparations of cod liver oil, in forms less offensive than the natural, may be resorted to. During the usually chronic progress of the case, a great variety of measures may be indicated, and many changes made, and often a reversion to some discarded measure may be advantageous. Still, while neglecting no measure which may be of service to our patient, it is scarcely necessary to keep them constantly under treatment, but, when in a comparatively fair state of health, they may be allowed a remission of treatment, and merely pursue a regulated diet, &c. Not, however, is this advised on any theory of the necessity for these intervals in the administration of digitalis, for in some cases this is found impossible, and it is positively demanded by the patient: indeed, the so-called cumulative action is beneficial in dilatation, though it is quite likely to produce poisonous symptoms when persistently administered in hypertrophy.

CHAPTER VI.

Affections of the Endocardium:—Acute Endocarditis—Ulcerative Endocarditis—Chronic Endocarditis.

Acute Endocarditis.—In describing the affections of the endocardium, some difficulty is experienced in drawing a line betwixt acute endocarditis and the more chronic process. The acute runs on into the chronic malady by a gradual process which leaves no marked division-line; but it is perhaps most desirable to describe as acute endocarditis what changes are usually found at the time, and within a short time after the acute inflammation, and as chronic endocarditis, the more gradual changes which are produced in time.

Acute endocarditis is, as its name implies, an acute inflammation of the living membrane of the heart, which is continuous with the endarterium, with which it is homologous. This inflammation of the living membrane of the heart is chiefly associated with acute affections, in which the blood itself is altered, as in acute rheumatism, Bright's disease, pyœmia, septicœmia, the acute exanthemata, and typhoid fever. It is thus found along with conditions of blood-poisoning with ostensible, or rather material agents. Whether these act as direct irritants to this serous membrane, or not, is not ascertained.

But we cannot feel surprised that we should have endocarditis when we have poisoned blood rushing through the heart, or that the worst form should be found in pyœmia, where Virchow has found the blood to have an acid reaction. In the most common forms of endocarditis, seen in acute rheumatism and chronic Bright's disease, the blood is also laden with an acid poison (lactic and uric acid), but not to the extent to alter its reaction. But it would appear that when the blood is so rendered less alkaline, we have a

marked tendency to endocarditis. But how it is brought about we have as yet no evidence: it may lie in the lessened alkalinity, or in the positive presence of acid blood poisons. So common is it in acute rheumatism, that Bamberger has calculated it to amount to 20 per cent. of all cases, a somewhat high percentage, probably due, in some degree, to his purely expectant treatment.

Pathological Anatomy.—The first stage is that of injection-redness, where the endocardium is injected, with points of branch-like vascularity. This is rarely seen, in consequence of the fatality of endocarditis lying rather in its consequences than its immediate action. This inflammatory injection must not be confounded with the mere staining of the endocardium with the colouring matter of the blood, a *post mortem* occurrence which not unfrequently happens, and where the endocardium is somewhat dull and of a dark red, or almost crimson, colour. The inflammation is not effusive, but belongs to what Virchow calls "parenchymatous inflammation" or the development of young cells without fluid. The serous membrane swells and is thickened, especially the free edges of the valves, and contains these soft young cells in its substance and beneath it. Among these cells are fine fibres and spindle-shaped cells. On the surface of the endocardium are found vascular growths, villi, often in small aggregations, giving the surface a warty appearance. These again must not be confounded with the mere fibrinous adhesions to the surface of the valves from deposit of the fibrin of the blood. These fibrinous vegetations, so called, are not villi, but mere strings of fibrin, and are usually found on that surface of the valves over which the current of blood rushes. The fibrinous deposits are far more apt to be detached and form emboli than the villous excresences which are the result of the endocardial inflammation. The endocardial inflammation is usually general, and is accompanied by more or less inflammation of the muscular structure beneath it. This myocarditis may extend some depth into the muscular structure, and by weakening it and altering its consistency, lead to bulging and the formation of a true ventricular aneurism. Endocarditis is often found along with pericarditis, or rather

they both take their origin ordinarily in the same conditions, and the intervening muscular structure is usually involved. The musculi papillaries are usually affected not only by the inflammation spreading over their surface, but also by their structure becoming involved. This leads frequently to irregular action in them, and to insufficient closure of the valve, especially during the early stages of the attack. Indeed, in acute rheumatism, irregular action of the musculi papillaries is not uncommon without any evidence of endocardial inflammation remaining.

As to the further progress of endocarditis to avoid repetition, it will be considered under chronic endocarditis.

Symptoms.—Endocarditis often runs its course without giving any evidence of its existence, except such as is furnished by auscultation. In acute rheumatism this statement contains no inherent improbability, as the patient's attention is fully occupied with the painful joint complications. That there should be no pain is not so unlikely, in consequence of the slight nerve supply of the endocardium, for this is nearly, not wholly, wanting. This accounts for the absence of pain in endocarditis, and, for a similar reason, there is little pain connected with pericarditis itself. There is no tenderness on pressure unless there is also myocarditis. At first the heart's action is somewhat excited, and the pulse is full, but this is only during the irritant first stage of the inflammatory process. When the inflammation has affected the muscular structure of the heart, there is evident loss of propulsive power, though there may be excited action of the heart and palpitation. Indeed, it would be somewhat opposed to what we know of palpitation if it were not present during this period of cardiac impairment. But this evidence of laboriousness is usually present, and the heart's inability is detected by the compressible feeble pulse which characterises the later stages. The inflamed valves being soft and thickened, do not produce so clear a sound as when in health. From this and the defective muscular condition, the first sound of the heart is muffled, dull, and wanting in clearness. This sound, too, is usually further masked by a murmur at the apex due to mitral regurgitation. This may

arise from valvular insufficiency, but is more probably due to irregular action of the musculi papillaries, which sometimes causes a murmur when no evidence of endocarditis is furnished, then or after. If, then, this irregular action is found without inflammation, it is more than probable that it is in action when there is. And, on the other hand, it is difficult to account for valvular insufficiency from change in the valves themselves, as this distorting and mis-shaping action of endocarditis is one of its later and not its earlier actions. The murmur may, however, arise from old standing valvular disease, and when so occurring, is decidedly troublesome from a diagnostic point of view. There are, however, points by which it may be diagnosed, approximately, at least; thus, when old standing mitral disease is present, there is old standing enlargement of the right side of the heart and accentuation of the pulmonary second sound.

As, however, mitral insufficiency, from the irregular action of the musculi papillaries, would soon make the diagnosis more complicated, it is desirable in all cases of acute rheumatism to examine the heart carefully at the first visit, so as to prevent any after confusion. For this mitral insufficiency soon leads to accentuation of the second sound at the pulmonary orifice, and to excited action of the right ventricle. The right ventricle, then, being distended, gives an increased breadth of cardiac dulness on the right side, and thus a condition of right side enlargement is simulated. The general symptoms of this condition are general malaise, anxiety at the precordia, tendency to delirium, and the general feverish symptoms. When there is mitral regurgitation, the pulse is often irregular in volume, and must be distinguished from the irregularity in time of the heart when enfeebled by the accompanying myocarditis. When the attack is more advanced there is weak feeble pulse, difficulty of breathing, congestion of the lips, and indeed the general symptoms of venous congestion.

The temperature is increased, and may show variations betwixt the morning and evening temperature of two or three degrees. The temperature is not commonly above 103° or 104° in the evening, but in consequence of the occur-

rence of acute endocarditis with acute rheumatism, it is difficult to fix a temperature as that of the endocarditis. The same holds good in the acute exanthemata pyœmia, puerperal fever, and the sœptic conditions along with which endocarditis is found.

Prognosis.—The prognosis of endocarditis is usually good. When occurring with acute rheumatism it is rarely or never fatal, and certainly not directly fatal. In Bright's disease it is also, in itself, not a source of immediate danger. In puerperal fever, in pyœmia, and sœptic conditions, the prognosis lies rather with the conditions than the endocarditis, and is very bad. The prognosis of endocarditis is altered by, and depends on, embolism. This complication is not rare, and may occur in any case. It depends on the washing loose into the current of blood one of the fibrin-deposits found along with endocarditis. Probably the inflamed surface attracts the fibrin of the blood more than uninflamed endocardium, and these form on the surface of the valves, over which the blood rushes, and are thus liable to become detached by the blood current. As the endocardium becomes inflamed chiefly over the mitral valve, and less commonly the aortic valves, and still less rarely the valves of the right heart, except in intra-uterine life, when the right heart is affected more than the left, these vegetations are chiefly located on it, and when so located may give rise to a presystolic murmur as the blood rushes over them. Thus, when detached, they pass into the general arterial current and float along till arrested in some artery which becomes too small to allow of their further passage. Here the embolon lodges, and cuts off the circulation, and results in necrosis of the part whose blood supply is thus arrested. The most common situation in which an embolon is arrested is the spleen, next the kidney, and after that the left middle cerebral artery. When then in endocarditis there are rigors, and evidences of any of these three occurring, as in spleen embolism, pain over the spleen, tenderness, and swelling; in the kidney, albuminuria, or pain over the loins, &c.; and in the brain, hemiplegia, aphasia, &c., there is ground for supposing that an embolism is formed. The embolon may lodge in the femoral artery, in the

coronary circulation itself, in the liver, mesenteric artery, and small ones even in the artena retinæ centralis. These embolisms and their consequences form the chief danger in acute endocarditis arising from the endocarditis itself.

The treatment of acute endocarditis will depend much on the affection with which it is associated. If with rheumatism, it is difficult to say, but this will be considered in the treatment of pericarditis, to which the reader may refer. In pyœmia and these conditions little but stimulants, &c., can be prescribed. In the acute exanthemata and in Bright's disease it will follow the treatment of the general conditions. There are no special means of affecting endocarditis, and bleeding, leeches, and the administration of mercury by inunction, and otherwise, against which Niemeyer is energetic, are probably more decidedly harmful than anything else. The same may be said of cold applications to the precordia. The chest should be kept warm, and be as little exposed in examination as possible. When there is failure of the heart, and defective circulation, digitalis may be administered.

Ulcerative Endocarditis.—Ulcerative endocarditis is a malignant form of endocarditis, not, however, apparently associated with any special zymotic or sœptic condition. It is common, however, under conditions of grave blood-poisoning, as in pyœmia, puerperal fever, &c. As it has been especially studied by Germans, and mostly in Berlin, the reader must pardon my giving an abbrievation from the account given by von Dusch (Lehrbuch von Herzkrankheiten).

This form of endocarditis commences with redness and injection of the tissue beneath the endocardium, which is swollen by a parenchymatous infiltration of connective tissue elements. There is also a free exudation from the surface, which is washed away by the blood stream. The epithelium is removed and the surface is dull, no longer smooth, but as if covered with fine felt. From the connective tissue arise white gelatinous mucus-patches, which appear on the surface of the valve as white granulations, having a greyish-red shade. These undergo a fatty degeneration, with a dissolving of the connective tissue, and are washed away little by little in the blood-current, and form superficial ulcers. These

are accompanied by greater or smaller collections in the valves which force up the endocardium and perforate it in spots, forming a purulent myocarditis. In other cases there is fatty destruction of the connective tissue with formation of pus in fine, small, yellow points. These pass on to a molecular necrosis of the endocardium with destruction and the formation of ulcers. This may lead to ulcerative destruction and tearing of the valves. When this occurs at the basis of a valve, it may cut loose a portion of the valve which floats freely in the blood: it has been known to sever a portion of the demolished valve, which has been washed away by the blood-stream. It also may destroy either the tendinous or muscular portion of the musculi papillaries, or it may also perforate the aorta valves and open the pulmonic and systemic circulations into each other. In other cases by perforating one surface of a valve, it may lead to aneurism of the valves; and where there is ulceration extending into the muscular structure of the heart, ventricular aneurism is formed.

Acute ulcerative endocarditis is seen either as a general blood-poisoning with typhoid symptoms and adynamic fever, or under the cover of the symptoms of pyœmia with the heart symptoms in the back ground. It is accompanied not uncommonly by other acute affections, as pericarditis, myocarditis, pneumonia, or pleurisy, and by numerous metastases. In most cases there are initial rigors with hot fits succeeding them, and at first these may have regular intervals, daily, or more frequently, resembling an intermittent fever: or the intervals may be irregular. Death may take place during the prolonged initial rigor. These rigors are succeeded by fever, and perspiration which gives no relief. In time the fever becomes more continuous, with slighter rigors. At the commencement there is vertigo, headache, great muscular weakness, and prostration. The pulse mounts up to 130 or 150, sinking sometimes suddenly to 80 or 90, and small and irregular. The temperature is 107°, the tongue dry; there is delirium, a drowsy condition, and stupor. There may be sudamina, and often is observed an almost roseolar exanthem, sometimes even papular or pustular, with more or less

general ecchymoses in the skin. The appetite is lost at the commencement, there is vomiting and diarrhœa with algor and cramps in the calves before death. In many cases there is jaundice from blood alteration, with metastasis to the liver, or parenchymatous inflammation of it. There is also enlargement of the spleen, with tenderness over it and the liver. The urine is scanty, saturated, and often coloured with bile, and frequently contains albumen. The patient may have oppression and pain in the præcordia with marked dyspnœa, choking, and orthopnœa, or there may be no subjective symptoms. When combined with pneumonia or pleurisy there are pains in the side, cough with frothy expectoration, blood-tinged, and the physical signs of these affections.

Among the objective symptoms are a systolic blowing murmur, or it may be diastolic: the heart's sounds may be obscured by exudation in the pericardium. On percussion there is general increased heart dulness. Death commonly occurs from the adynamia passing into coma, or is sudden from the tearing off of the valves and chordæ tendineæ.

Prognosis.—The prognosis is very unfavourable indeed, and the progress of the case to its termination is rapid.

The Diagnosis.—This affection is closely allied in symptoms to typhoid fever, in the enlargement of the spleen, the diarrhœa, roseola, delirium, and stupor, and is distinguished from it by the prolonged rigor and intermissions, and by the positive evidence as the case proceeds, and the intermissions are lost, of the presence of mischief within the heart.

The treatment has consisted of stimulants, ammonia, wine, camphor, and musk, and the administration of quinine in large doses in combination with opium; the subcutaneous injection of morphia, and the external use of ice and salt.

Chronic Endocarditis.—The pathological changes described under acute endocarditis may pass on into a slower chronic condition, which, however, commonly occurs without any initial acute inflammatory action. The acute inflammation, with its thickening of the endocardium and its villi over its surface, passes away, and a condition of much less vascularity takes its place. The injected and redened endocardium

becomes paler, and ultimately an opaque white membrane, or may simply lose its vascularity and resume its natural appearance with the distorting action going on beneath its surface. But, soon, all characteristics connected with acute endocarditis pass away, except it be in a more rapid destructive process than when purely chronic, but presenting no naked eye changes to distinguish it. The chronic process is a distinctly parenchymatous inflammation, slow, and having none of the characteristics of inflammation, except it be sometimes "swelling." The development of connective tissue slowly, with its contracting, distorting action, is the characteristic of chronic endocarditis. At other times there is distinct increase in bulk, owing to a more free development of cell elements, especially in the free edges of the valves, which become rounded, hard, and incompressible. On the semi-lunar valves it may take the form of festoons along each valve. But any increase in bulk is ultimately more or less reduced by the contracting process so peculiar to connective-tissue elements.

It may be convenient to describe in turn the process as it goes on in the auriculo-ventricular and semi-lunar valves. In the mitral valve are the changes due to chronic endocarditis most frequently seen. They may proceed in two directions towards drawing the valves in the direction of their musculi papillaries, or in a direction towards each other, by the agglutination of their free surfaces. In the first case the process goes on not only over the valves themselves, but along the endocardial covering of the tendinous cords of the musculi papillaries. This process is frequently confined to one valve only with its cords, and by preference the valve of the fixed wall. In the mitral it more commonly attacks the valve of the fixed wall and the small sheet-like patch of endocardium, with fibrinous threads glittering through it, which belongs to the fixed wall valve of the mitral, and extends into the aortic valves on that side. The contracting process may draw the free edge of the valve down to the points of the papillary muscles themselves, or the action may lead to withering and absorption of the musculi papillaries themselves, and the valve is fastened down to the muscular

wall of the heart by the tendinous cords merely, and is so generally contracted that the tip of a finger only can be inserted betwixt the wall and the valve. This last action is not uncommon in the mitral, and is perhaps the most common pathological process in the tricuspid.

At other times the chronic process gradually approximates the free edges of the valves, commencing from their extreme edges and gradually gluing together the free edges until ultimately all trace of two valves is lost, and there is either a firm curtain, merely perforated in the centre, dividing the ventricular and auricular chambers, or at other times the process draws the two valves with their cords and muscles together into a perforated cone, extending into the ventricle, with a rough edged perforation into which a finger cannot be passed. The closure is sometimes so advanced, ere death, that the orifice is merely a slit, termed "a button-hole" mitral. Usually behind this, the inflammatory process has spread over the surface of the auricle and converted the endocardium into a firm white opaque lining, so rigid as to prevent the auricle from collapsing: this process extends into the pulmonary veins, and is due probably to the stretching of the parts from the obstruction in front offered to the flow of blood; in this it is allied to the atheromatous changes in the lining of veins when distended by an artery opening into them. This stenosis produced by chronic endocarditis is confined to the mitral valve, and stenosis is yet unknown in the tricuspid. Changes in the mitral valve produced by chronic endocarditis are not uncommonly accompanied by tricuspid insufficiency from a similar cause.

In the semi-lunar valves this chronic inflammatory process becomes very frequently a true chronic valvulitis, not affecting any other portion of the heart. It is an insidious complaint, without subjective symptoms of its own, and slowly works the destruction of the valves, causing either stenosis, or simple insufficiency, and commonly a combination of the two. Usually when seen at an early stage, the free edges of the valve are thickened, round, and cordlike, yet capable of almost complete closure. The corpora arantii soon become obliterated and lost. The contracting action often, acting on

this cord, diminishes the size of the valves, so that on the aortic recoil they can no longer approach each other. The tendency here is to contract each valve individually, and draw them towards the walls of the aorta, and thus, while offering no obstruction, or little, to the forward rush of blood on the ventricular systole, to offer no obstruction to the backward rush of blood on the arterial recoil. Where this tendency to contraction of each valve is marked, the case runs its course rapidly. At other times there is fulness of the free edges of the valves, with somewhat of shrinking, so that the edges no longer approximate and insufficiency occurs, and this is accompanied by thickening of the valves generally and more or less rigidity, so that there is also an obstruction, and systolic murmur, from the rigid valves not bending back before the blood-current. At other times there is rigidity of the valves gradually becoming more and more perfect, with agglutination of the edges of the valves until the orifice is distinctly narrowed, so as to offer great obstruction to the flow of blood through the valve either forwards or backwards. This may proceed to great length, ere death, and has gone so far that a probe could scarcely be passed through the aortic orifice. At other times this rigidity fixes the valves before such contraction has been reached, and from these rigid free edges grow tubercles of chalky matter, extending over the orifice, and still further impeding the blood-current. At other times it may glue together two valves, and these, stretching across, oppose both flow and regurgitation. From the convex side of these valves being presented to the blood current from the ventricle as long as any flexibility remains, it is in favour of the normal current, while the concave surfaces being presented to the backward rush of blood, regurgitation is opposed. In each case it is prognostically important to ascertain how far the lesion bears the character of obstruction or insufficiency.

Valvulitis is not always confined to one valve, and aortic disease often becomes complicated by the process spreading to the mitral valve, and *vice versâ*. It may sometimes extend from aortic valves to pulmonary, but this is scarcely direct extension through the arterial coats. The pulmonary valves

themselves are rarely affected, especially when not subjected to increased strain from pre-existing disease of the left side. This corroborates the view that valvulitis usually arises from the force with which the valves are closed, aided, no doubt, by favouring dyscrasial conditions.

When this proliferation of connective tissue elements has existed some time, it undergoes degenerations itself. This may be by excessive deposit of earthy salts in it, or by absorption of the other elements, the salts remaining. There remains a petrified condition of the valves, often with cretified masses, the remains of outgrowths, attached to the diseased valves. These last very commonly line the diseased valves, both mitral and aortic, and make the edges rough and irregular, aiding much in the production of loud murmurs, especially systolic. At other times this calcification goes on with certain connective tissue corpuscles enclosed in it, and then a process, closely resembling petrifaction is inaugurated, the valves being hard and stony. This is probably what older writers described as "ossification of the heart," rather than any development of bone within the walls, as is normal in some animals, and commonly seen in the heart of an elderly ox or cow; though this does occasionally occur. This ossification, or rather petrifaction, is persistent. In other cases the new tissue undergoes fatty degeneration and the formation of cholesterine scales, as in the degeneration of an atheromatous arterial patch.

Frequency in Valves affected.—This is important diagnostically, as well as interesting pathologically. When chronic endocarditis is a prolongation of acute endocarditis, which was co-existent with acute rheumatism, the mitral valve is almost invariably the one affected. Others may be affected along with it, as the aortic valves, and more rarely again the tricuspid; the pulmonary probably almost never. But in advanced life, when a chronic valvulitis is common at the aortic orifice, the order is changed, and the mitral is less commonly affected than the aortic, but often becomes affected secondarily to the aortic valves. When there is old standing disease of the left side of the heart or of the lungs, and there is dilatation of the pulmonic vessels with enlargement of the

right side of the heart, then we get valvulitis of the pulmonary valves. This is not commonly seen, as here is often tricuspid endocarditis, and the accumulation of evils carries off the patient before the pulmonary valvulitis becomes marked. This tricuspid inflammation is also due to the enlargement of the right ventricle and the more forcible closure of the tricuspid. Thus tricuspid lesions are not rarely found in connection with mitral lesions, but very rarely as a primary lesion, and then usually with old-standing disease in the lungs, or as remains of intra-uterine affections.

Elaborate tables have been formed of statistics of the comparative frequency with which the valves of the heart are affected. The only one at hand is from von Dusch, but will illustrate the relative frequency as well as a larger collection, for the relative frequency is, within certain limits, pretty regular.

Willigk found the relative frequency of valvular affections, in his experience, to be mitral disease 36; aortic disease, 22; tricuspid, 8; pulmonary, 2. Flint found mitral disease 40; aortic, 37; of both combined, 14; tricuspid, 4; Cockle, mitral disease 90; aortic, 71; both combined, 17.

Œtiology.—These tables indicate the probable cause, as well as the relative frequency. Mitral disease is most common, being the result of acute rheumatism, the great provoking cause of endocarditis. Next comes aortic disease, associated rather with advanced life, and thus with atheroma of the aorta, and frequently with gout. Then comes the union of aortic and mitral disease, in most cases probably due to the effect upon the mitral of the aortic disease, in producing hypertrophy of the ventricle and more forcible closure of the mitral valves, and thus disease in them. Then comes tricuspid disease, where the tricuspid is closed under greater force by an enlarged right heart; and, lastly, the pulmonary, bearing the stress of the gorged pulmonic circulation in left-side disease, and then not often found diseased to any detectible length in life, and still more rarely the subject of primary disease. It would appear from this that acute endocarditis is the greatest excitant of chronic endocarditis, and, next to it, forcible closure of the valves. This is the cause

of disease of one valve following disease of the one in front, and disease of the right side of the heart disease of the left. We may here review some of the exciting causes of endocarditis associated with strain, and this is primarily seated in the aorta. The two great causes of aortic disease, are gout, in the aged, or other causes of atheroma, but gout, or perhaps rather chronic renal disease, certainly first; and, in the young, violent efforts, especially such as implicate the whole muscular system, and are connected with use of the arms. This last form of aortic valvulitis in young persons, or those of middle age, is very commonly seen in "strikers," hammermen, colliers in thin beds of coal, boat racers, gymnasts, &c. It is extremely common among strikers, and was first carefully observed in connection with them. These men have to exert themselves violently during the brief time that heated iron remains sufficiently hot to be worked. This is not long, not more than one minute, or one minute and a half, and during the process of heating the striker is resting, but, if then examined, he will be found with intense vascular excitement. This becomes somewhat modified ere the next call, which is not far distant. This continues for at least ten hours in each day, and is steadily pursued as an occupation. None, of course, but strong and powerful men aspire to this position, and their efforts are stimulated by a natural rivalry. On examining this form of labour as a cause of aortic valvulitis, one peculiarity is observed about it. The labour with the arms necessitates a fixed position of the muscles of the body generally to enable the striker to deliver his blows with precision, and with greater force. This affects the circulation in two ways. 1. The muscles of the body largely cross the arteries, and, when in action, constricts them and impede the flow of blood through them; this action is termed by Wardrop "the musculo-cardiac function," and he thought it tended to keep the ventricle full of blood. This it most unquestionably does, for the ventricle cannot empty itself completely into the aorta, when the onward progress of the blood is obstructed by these muscles crossing them. 2. In this condition of muscular activity the entrance of the blood into the muscles is impeded, and thus another great obstacle

offered to the arterial flow, during this period of general muscular activity. From these two causes combined the blood flows slowly into the arterioles of the body, and great resistance is offered to the flow of blood. This soon brings out hypertrophy of the left ventricle, and then the blood is forced into the aorta under greater pressure, while its progress is hindered, and thus the aorta is excessively distended and recoils forcibly, driving together the aorta valves under unwonted pressure. This in time evokes valvulitis, with its changes. But this obstraction to the flow is not all the mischief in these cases. While the muscles obstruct the arterial flow they aid materially in accelerating the venous flow, contracting as they do in the direction of the trunk. Thus, while the blood is squeezed out of the muscles by their action, the blood in the veins is being driven towards the heart. This leads to its accumulation in the great veins, which are gorged, they fill the right heart under increased pressure, while the pulmonary flow is impeded by the difficulty with which the left ventricle disposes of its contents: then comes hypertrophy of the right side of the heart. The blood is now driven into the left ventricle under increased pressure, and dilatation is added to the already existing hypertrophy; further hypertrophy is then elicited and a condition of dilatation of the left ventricle with decided hypertrophy is established. From this again a larger quantity of blood is thrown at each systole into the aorta, and a condition of over-distension of it kept up: this adds further to the strain and consequent valvulitis.

This is the ordinary history of aortic disease in the striker, who often complains of palpitation, &c., ere any sign of valvular mischief can be detected. This muscular inability is in time overcome by hypertrophy, which protects it, but helps to hasten on the destruction of the aortic valves. A precisely similar course of events goes on in the collier, working in thin beds or seams of coal. Here a very constrained position is necessitated, which requires for its maintenance general muscular action, while the collier exerts himself to the utmost on each "shift," resting in the meantime. Here the same consequences are seen as in strikers.

There is also an approach to this in the general muscular effort demanded from the oarsman in boating, and heart disease is developed in them frequently, but to a less extent than in the above cases, because their efforts are confined to training-times, that is, the very desperate efforts, and are limited to a few years ordinarily. A similar history is associated with gymnastic efforts, but commonly the palpitation and other signs of the preliminary stage of muscular trouble deter from their efforts those with whom it is a matter of choice, while the striker and the collier must labour on. This effect of the muscular debility is often very valuable in inducing the followers of these pursuits to abandon or modify these efforts, and thus the deleterious efforts are given up before the latter stage of decided and marked aortic valvulitis is produced, while in those where muscular growth is good, this stage of compensatory hypertrophy is reached, and mischief inaugurated, which manifests itself some years after, when its cause is usually forgotten. That valvulitis should thus hesitate and pause in its progress is not surprising if the provoking cause be in abeyance, and often after their University days these violent boating efforts are given up for a comparatively inactive life in the Church's service, and then the mischief lingers in its career.

The effect of gout, or, rather, chronic kidney disease, produces a very similar effect on the aortic valves. The effect of retained urine salts in the blood has been shown by Professor Geo. Johnson, Dr. Kelly, and others to consist in hypertrophy of the muscular tunic of the arterioles. These contract, and oppose the onward progress of the blood, hypertrophy of the left ventricle is induced, and, when induced, the aorta is filled under a stronger ventricle, and betwixt the hypertrophied ventricle and the hypertrophied muscular coat of the arterioles the aorta is over distended, and its recoil closes the aortic valves violently, and aortic valvulitis ensues. But this subject will be discussed at length in Chapter XIII. For the further progress of aortic valvulitis and its consequences the reader must refer to the Chapter (VII) on valvular lesions.

Thus we see that aortic valvulitis is associated with

strain, with closure under excessive force, when an aorta is over distended betwixt an hypertrophied left ventricle and an obstruction offered to the blood current. So, too, mitral disease may be found along with aortic disease, but usually not till aortic regurgitation has dilated the heart and produced great hypertrophy, and thus the mitral valve is closed under greater pressure. The endocarditis may also spread from one valve to the other, in addition. Mitral disease often follows, but rarely precedes aortic regurgitation.

So, too, we see disease of the right side following left side disease, and rarely otherwise, except when chronic lung disease opposes the flow through the pulmonic circulation, and right side enlargement is developed. Thus the pulmonary valves are rarely found affected, and not so often the tricuspid, in consequence of the patient's death from the consequences of venous congestion and impaired circulation. Still we have enough of evidence to show us that valvular endocarditis is associated with over-strain of the valves,* and if additional evidence were required to substantiate this view it would be found in the endocarditis and thickening found in the left auricle when subjected to over-distension in mitral stenosis; and in the endarteritis found at points where the endarterium is stretched and exposed to strain; and often found along with aortic valvulitis where the aorta is continuously over-distended; and, finally, in the atheroma in a vein which is over-distended by an artery opening into it.

Chronic endocarditis may result from other, as yet less known, conditions, especially in dyscrasial conditions, but next to acute endocarditis certainly over-strain is the great provoking cause.

Symptoms.—The symptoms (objective) of chronic endocarditis are nil, nor are the subjective symptoms any more, except by its effects upon the valves. There is a very limited nerve supply; indeed, its existence is rather conjectural than

* This subject is well shown in some cases given by Dr. Clifford Allbutt in his pamphlet on "The Effects of Overwork and Strain on the Heart and great Bloodvessels." Reprinted from St. George's Hospital Reports, 1871. Macmillan and Co.

demonstrated in the lining of the heart and blood-vessels, and affections of them do not give rise to pain, or other symptom, to indicate their existence.

Prognosis.—The prognosis will depend on the valvulitis and its consequences, and must be followed out in the chapter on Valvular Lesions.

Treatment.—The treatment resolves itself into a removal of the exciting cause, where practicable. The abandonment of severe athletic exercises when an undesirable effect is being produced on the heart or vessels, and, in those who must labour, the adoption of some new form of labour not necessitating these efforts, is desirable. It is of great importance that these causes of endocardial disease and its terrible consequences be fully recognised. The distance of the danger, and the entrancing nature of the voluntary pursuits, make their votaries somewhat inclined to be sceptical about the consequences, and to attribute the ultimate consequences to some other cause in action in the interim; but this is not wise, and the sooner an intelligent recognition of the consequences of these muscular amusements now so largely in fashion, the better for those engaged in them. Physicians' advice has been rejected by them rather rudely; but they must remember that we can have no interest in checking these peculiar sports; indeed, our interests lie the other way; and we probably enjoy the spectacle afforded as much as other people do.

The more especial treatment must be followed in the treatment of each lesion, and in the chapter on Treatment.

CHAPTER VII.

Valvular Diseases—Aortic Obstruction—Aortic Insufficiency — Combination of — Mitral Obstruction — Mitral Regurgitation—Combination of—Diseases of the Pulmonary Valves—Tricuspid Insufficiency.

When then this process of alteration of the tunica intima, so called atheroma or endarteriitis deformans, has affected the lining of the aorta and heart, including the surface of the valves, we find that the morbid process may distort or deform the valves so as either to cause them to offer a more or less decided obstruction to the flow of blood through them, or to be more or less unequal to prevent its regurgitation, that is, they become insufficient to fulfil their function. These conditions are commonly enough combined in various degrees, but usually one or other condition is sufficiently preponderating to entitle us to call it that disease with the other, for instance, in aortic disease we may have both forms of disease united, and we call it aortic obstruction with regurgitation where the stenosis preponderates, and aortic insufficiency with stenosis when the regurgitation preponderates. So, too, in the pathology and sequelæ, whichever preponderates will be followed by the sequelæ more peculiar to it, modified of course by the amount of the disease combined with the chief affection. This is equally the case with diseases of the mitral valve, as regards the diagnosis which is often rather difficult, and is much aided by the character of the pulse, a subjective symptom of the greatest value in aiding us to form a correct diagnosis in valvular diseases of the heart. There is no doubt some difficulty in learning to distinguish the different valvular lesions of the heart, and their accompanying conditions, but this is much magnified by the fears of the student and want of confidence in his own powers, and perhaps to some extent by writers not keeping the

students' difficulties sufficiently before their eyes, and compiling their book accordingly. A little true thinking over the necessary origin of certain sounds heard, and the effect the lesion must have on the parts behind it; careful examination of the healthy heart, and of some well marked lesions in the hospital, and attention to the remarks of his teacher, will soon enable the student to distinguish a lesion as clearly as most men; but if the student will attend to tricuspid lesions, combined murmurs and lesions, &c., before he is in possession of his alphabet and gets muddled, it is very possible that he has only himself to blame. But if he learns his alphabet soundly and learns to spell the simpler words first, in time will he not only find himself equal to spelling out the most complex combinations, but will feel a pleasure in his own power so to do. This power acquired, no diseases admit of so close diagnosis as heart diseases; mistakes are undoubtedly made, and by the best men, but human nature is fallible and liable to err from haste or carelessness; still heart diseases can be disgnosed with a certainty which does not belong to abdominal diseases for instance.

The time at which a murmur is heard, in relation to the heart's sounds, its point of maximum intensity, to some extent its character, the amount of hypertrophy and dilatation, or either, and its peculiar pulse, are usually sufficient with intelligent thought to fix the seat of the lesion, and its amount. In aiding in forming a prognosis and guide to the development of sequelæ and the appropriate treatment, it may be well to insist on three divisions. When the aortic orifice alone is affected, and the mitral remains perfect, we have little or none of the subjective symptoms due to obstruction of the circulation; when the mitral is involved, we have lung symptoms, or rather chest symptoms necessarily evoked; when the pressure of obstruction is affecting the right heart, and producing decided dilatation, and still more if the tricuspid is involved, we have general venous stagnation and its consequences.

Aortic Obstruction.—The first disease then under the first of these three divisions is aortic obstruction or stenosis. This is not an uncommon form of heart affection, especially

in advanced life, and is usually well marked, but not uncommonly associated with more or less regurgitation. It is always found with hypertrophy, because the ventricle is not filled under undue pressure, the great cause of dilatation; its pulse is characteristic.

The pathological changes which give rise to obstruction at the aortic orifice at least, are invariably the result of endarteriitis deformans, or atheroma. At first the valves are somewhat thickened and rigid, and fold backward when the blood column is driven through them on the ventricular systole. This produces a sawing murmur, which is systolic. The valves become more rigid and thickened, opaque and contracted, often atheromatous vegetations spring from them and almost close the orifice entirely. The extent to which this goes on is often simply surprising, and life has been maintained until a bristle could scarcely be passed through this opening. This obstruction necessarily is accompanied, or rather followed, by changes in the muscular walls of the heart. This is invariably hypertrophy, and usually of the purest kind. The obstruction is not one which comes on suddenly, but rather in the most gradual manner, and consequently there is no primary formation of dilatation, as happens when the obstruction offered is rapid in its formation. Simple hypertrophy is the muscular condition associated with this lesion. This affection is not commonly associated with other valvular lesions, and is not usually accompanied by the general symptoms of heart failure until a very advanced condition of stenosis, or of fatty degeneration of the walls is reached. This last is almost sure, sooner or later, to follow from imperfect filling of the aorta, and subsequent imperfect circulation in the coronary vessels. The affection is essentially chronic in its character. It is, too, most commonly found in advanced life, so that it contrasts with the frequency of mitral disease in the young. It is, indeed, a disease of middle and advanced life, and is essentially associated with the atheromatous process.

Diagnosis.—On inspection and palpation nothing is to be discovered more than a distinct pulsation at or below the sixth rib, regular, defined, and powerful. There is in fact true

hypertrophy of the left ventricle; rarely, or never, mixed with dilatation unless there be also regurgitation, or the hypertrophy is undergoing degeneration. Percussion, too, shows an increase of dulness over the left apex and below, corresponding to the apex beat. Auscultation is the great means by which we distinguish positively the existence of aortic obstruction. Firstly, in connection with the signs furnished by percussion, palpation, &c., we find the loud but muffled first sound of hypertrophy; while the blow on the chest-wall is distinctly communicated by the stethoscope. Then we find also a sound of a sawing character, an obstructive murmur accompanying and often masking, more or less completely, the first sound. This sound is heard with maximum intensity over the second right costo-sternal articulation, it can be followed up the aorta, along the carotids and subclavians, and even further. It is a comparatively loud murmur, and is heard over a large area, over the base of the heart, at the left apex, and at the ensiform cartilage. It thus simulates mitral and tricuspid regurgitation, and all murmurs heard with the first sound must be tracked to their point of maximum intensity. Without this many murmurs will be hastily set down as mitral or tricuspid, from the clearness with which they are heard over their areas, unless they be followed out.

The objective symptoms are as marked as the physical signs. The action of the hypertrophied heart with aortic stenosis is regular, usually slow, and rarely palpitating. There is the peculiar heaving, steady, character of pure hypertrophy. After severe exertion, or exhaustion, there is a tendency to intermit. The exhausted ventricle is taking a longer rest before it can meet that obstacle offered to the forward progress of the blood column. This halt is sometimes very prolonged. Consequently, aortic obstruction is one of the forms of heart disease associated with sudden death. That halt may be permanent. Aortic stenosis is thus liable to sudden death; while it is also more free from the subjective symptoms and consequences of obstructive circulation.

The pulse of aortic obstruction is small, on account of

the obstruction at the aortic orifice, hard and sustained from the hypertrophy. The pulse is hard, incompressible, and sustained, while it is small, and thin or wiry. It is not unlike the pulse characteristic of abdominal inflammation.

Aortic obstruction is liable to be confounded with some other affections, from the time and position of its murmur. In time it corresponds with mitral regurgitation. In time and position with aortic dilatation and aneurism of the ascending aorta. It would, perhaps, be more correct to say that these diseases are apt to be mistaken for aortic obstruction: and they will be considered as regards their diagnosis, &c., in the chapter on Diseases of the Great Vessels (Chapter XIV).

In time and position this murmur is exactly simulated by the anœmic bruit. In many conditions of anœmia, and especially in chlorosis, a murmur is heard in the heart exactly corresponding to aortic stenosis. The blood deficient in quantity, or depraved in quality (spanœmia), furnishes in its passage through the aortic orifice murmurs from vibrations in the semi-lunar valves.

The differences are, in anœmic bruit, first youth and sex, being usually in girls, the positive evidences of anœmia and debility, and a weak, short, compressible pulse; and wanting the characters of aortic obstruction, as hypertrophy and its signs and symptoms, and the sustained and compressible pulse; the murmur, too, is not communicated into the distant arteries, though in the neck it is simulated by the bruit de diable, the vibrations in the jugular valves; this can be arrested by pressure of the finger; the aortic murmur cannot be so arrested in the carotids.

From mitral regurgitation it may be distinguished by a variety of measures, as the position of maximum intensity, the conduction of the sound, the absence or presence of lung symptoms, &c., hypertrophy, as compared to hypertrophy with dilatation, and finally by the character of the pulse.

Characters special to Aortic Obstruction.	*Characters special to Mitral Regurgitation.*
Systolic, but heard with maximum intensity at second right costo-sternal articulation, and communicated along the arteries.	Systolic, but heard with maximum intensity at left apex, and in a line passing behind the nipple, or over the left auricle.
Rarely connected with lung symptoms.	Intimately connected with lung symptoms.
No accentuation of pulmonary second sound.	Accentuation of pulmonary second sound.
Pure and simple hypertrophy of left ventricle and well defined apex; beat usually powerful.	Mixed hypertrophy and dilatation; apex beat diffused, and not so marked in power.
Pulse hard, firm, wiry, and sustained; regular, and when not so regular in time, arrested or intermittent.	Pulse irregular in volume, more or less blood always escaping through the mitral valve. Not sustained or incompressible.
Tendency to sudden death, and absence of consecutive complications.	Tendency to linger on, with consecutive complications.

These points are sufficient to enable a diagnosis to be made betwixt aortic obstruction and mitral regurgitation.

The aortic obstructive murmur is often simulated, too, in fevers by the altered blood, but this is not likely to lead to any mistake; it is sufficient to merely mention it.

Prognosis.—The prognosis of aortic obstruction in the early stages, and when not very marked, is not bad as to duration of life: as to recovery, like all valvular lesions, it is hopeless. The condition of the muscular walls must form the foundation of prognosis, and when the ventricular systole becomes feeble, and more so if the feebleness be indicated by intermittency, the prognosis is becoming very bad. It is not a form of valvular disease commonly followed by dropsy and other consecutive changes, but, on the other hand, it is somewhat liable to genuine apoplexy, especially when combined with morbus Brightii. There is a decided risk of sudden death, especially if that ventricular chamber should be over-distended with that narrowed orifice in front, the over-distended ventricle may be unequal to the effort, and remain permanently in diastole. It is found associated with cerebral anœmia, especially in fatigue, and when the heart is exhausted: the prolonged halt, by cutting off the arterial blood supply to the cerebrum, may cause the patient to fall from cerebral anœmia. In one case that was remarkably well seen; and treatment of the heart effectually relieved the head symptoms.

Treatment.—The treatment of aortic obstruction is according to the rules laid down in the chapter on Treatment. Just one point must be alluded to. Much has been said and written on the subject of digitalis, slowing the heart's action, and allowing more time for the blood to flow through the narrowed orifice. This is erroneous; it is not a question of time; that would not alter matters; it is a question of increasing the driving power, the only means by which an equal bulk of blood can be driven through a narrowed orifice in equal time.

Aortic Regurgitation.—The next affection of the aortic valves is insufficiency. It is allied to aortic regurgitation both in situation, in causation (pathological), and in its freedom from the systemic consequences of venous congestion, until the mitral orifice becomes affected. This last is, however, more common in aortic regurgitation than in obstruction, as more causes are in action than the mere spreading by contiguity of atheroma from the aortic orifice to the mitral valve of the fixed wall or septum.

In aortic regurgitation the atheromatous process has rather contracted the semi-lunar valves than converted them into rigidity. In slight affection of this form of lesion we find the free edge of the valve thickened, cord-like, and contracted. In other cases the process has had rather an ulcerative tendency, and one side of a valve has given way. In other cases a violent effort has been known to tear down these valves. In fact, aortic regurgitation is essentially connected with effort. In the iron districts of England this form of disease is unusually frequent, especially among "hammer men" or "strikers"; but it is also found among colliers, and more so when there are thin seams of coal, and the collier has to work in constrained positions. It has been observed to be associated with labour involving great use of the arms, a fact not only curious but instructive in relation to the pathology of this affection. For the exciting cause of the low interstitial inflammation, with growth of connective tissue, is essentially the violence with which these valves are closed, or rather driven together by the backward rush of blood on the aortic recoil. When, then, recoil is increased,

so is the force with which these valves are closed. This is well seen in the condition of these strikers immediately after a "heat." He is panting with hurried respiration and excited action of the heart and pulse. During his brief exertion his muscles have been in violent action, and many of them cross the arteries, as Wardrop has pointed out (his musculo-cardiac function), and thus oppose the onward flow of blood. His chest is fixed in order to furnish fixed points from which the muscles work, and the whole frame is in a posture to enable him to deliver his blows with greater precision and force. We have then the muscles crossing the arteries and obstructing the flow of arterial blood, while their action is decidedly to propel the venous blood towards the heart. This tends to fill the great blood centres, and a partial hypertrophy of the heart commences. The ventricle, too, filled under greater pressure from the gorged pulmonic veins, is of full capacity, so that we find a strong and large ventricle with a large chamber acting against the obstruction offered. Even previous to the formation of aortic valvular disease, these men have often heart symptoms as palpitation, &c., showing that the ventricle is taxed. In time the hypertrophy is distinct. Thus we have a powerful heart opposed to the obstruction offered, and consequently great aortic tension, and the valves are closed violently. In considering the connection of this lesion with the use of the arms, we must bear in mind this point. When the labour is in the arms, the body must be kept in a certain fixed posture, and this entails general muscular rigidity. Almost all the muscles of the body are brought to bear on the maintenance of this postural attitude. Consequently, these rigid muscles offer an obstruction to the entrance of the blood into them, and thus a great obstacle is furnished to the arterial blood stream. This condition of general muscular action is not found in other forms of labour, and supplies us with the explanation of the connection of the form of valve lesion with labour entailing great use of the arms. Sooner or later this leads to such an amount of disease in the aortic valves, as to render them insufficient, and at the same time often a dilated and somewhat rigid aorta itself.

In connection with this explanation of the œtiology of aortic regurgitation, may be adduced some statistics collected by Bamberger. In 50 cases of aortic insufficiency, Bamberger found 38 men and only 12 women; though women do much of the coarser kind of labour in Vienna. Of these 50 cases 15 were under 30 years of age.

It is not meant from this that aortic regurgitant disease always takes its rise in excessive bodily exertion, but merely that that is a common cause: it is also connected with Bright's disease, endarteriitis deformans, and endocarditis.

When, however, the blood has once commenced to flow backwards through the aortic orifice, a new series of changes are at once instituted. The bulk of blood driven back by the powerful aortic recoil is added to that normally coming in from the pulmonic system through the mitral valve. By the combination of these two streams, the left ventricle is distended in its diastole by an increased bulk of blood driven in under increased pressure. The result of this, as shown in a previous chapter, is to dilate the heart, and hypertrophy is rapidly developed to resist this excessive distension. This goes on to a remarkable extent, and the heart becomes both dilated and hypertrophied to unusual dimensions the corbovinum. It may even reach down to the eighth intercostal space. This excessive hypertrophy with dilatation causes an unusual bulk of blood under violent pressure to be forced into the aorta and arterial system generally. This in its turn becomes distended and dilated, and in a little time atheromatous. This violent distension can be felt even in the anterior tibialat, the ankle, and has been seen by the ophthalmoscope in the arteria centralis retinæ. But this excessive distension is brief in its duration, and the blood escaping backwards at the recoil, it is cut short as it were. This is well shown in a sphygmograph tracing. The sensation to the finger is that of "balls of blood shot under the finger," a most characteristic expression. The disease proceeds in every direction, the aortic orifice becoming more insufficient, the aorta thickened, and the backward rush of blood both greater in bulk and driven back more violently, so the heart's chamber becomes more dilated and more

hypertrophied. This in its turn forces a larger mass of blood at each systole violently into the arteries, the vessels from this excessive distension become atheromatous. In the meantime this arterial distension gives the patient headache, vertigo, &c., bleeding at the nose, and when their structural integrity is impaired, the arteries of the brain are liable to rupture, and true apoplexy to happen. This result of increased arterial pressure is quite different from the apoplexy produced by impeded venous flow in consequence of heart failure. It is the direct consequence of dilatation and hypertrophy not connected with, and therefore not neutralised by, obstructed blood flow. The distension causes, too, pulsation of the abdominal aorta, in females menstrual disorders and increased loss of blood at each menstrual period, and even, according to Oppolzer, albuminuria from increased tension in the renal artery.

But this condition is not permanent, and the excessive hypertrophy does not persist. This has been most ingeniously pointed out by Mauriac. He has shown that the cardiac nutrition supplied by the coronary arteries is soon involved. The aortic recoil is the propelling power into the coronary circulation, and when this is no longer arrested in the backward direction by the aortic valves the flow into the coronary vessels is diminished. So we find that this hypertrophy is not long in undergoing tissue-degeneration. This is aided, too, by the fact that the atheromatous aorta and large vessels lose their elasticity, and do not recoil so perfectly. These combined causes soon affect the structural integrity of the heart walls, and the heart is less equal to resist the distension. It again yields, and dilatation recommences, hypertrophy to restrain it being no longer possible. The patient rapidly sinks, but as long as the mitral valve is unimpaired, there are not the symptoms of obstructed venous circulation. But the patient gets feebler, his pulse becomes hesitating, intermits, and finally ceases in diastole. Not uncommonly the mitral does become affected, and that by various means. The most common one is the extension of the atheromatous process from the aortic valves along that small sheet-like portion of endocardium which stretches from the base of the

aortic semilunar valves downwards, and forms the valve of the fixed wall of the mitral orifice.

The great dilatation of the chamber, too, may lead to the valves becoming incompetent, but, as Rokitansky shows, the valves and musculi papillaries ordinarily stretch with it, and so no insufficiency arises. But under these circumstances of excessive action of the ventricle, the valves and musculi papillaries, with their tendinous cords, are apt to become structurally modified, and this greatly encourages the extension of the mischief from the aortic orifice to the mitral valve. This involves the tendinous cords, and they become shrivelled and the valve imperfect. Traube has advanced a theory that the mitral valve becomes incompetent from atrophy of the musculi papillaries. This is produced in this way: the rush of blood backwards through the aorta puts the valve of the mitral of the fixed wall on the stretch during the diastole, when it should also rest along with the other muscular structures. During systole it is of course on the stretch, and this incessant action both deprives the musculi papillaries of their due rest and also of their nutrition. They atrophy, become incompetent, and the valve, no longer sufficiently restrained by them on systole, is washed back by the powerful ventricle. Thus, by one or other actions, the mitral valve often becomes involved in aortic regurgitation. When this occurs, the symptoms especial to aortic disease become modified by those symptoms of mitral failure and its sequelæ, which will be more especially considered in that section.

The *diagnosis* of aortic regurgitation presents no great difficulties, and the points of it are such as might be expected from its pathology. The signs are as following:— On inspection, a distinct pulsation over the cardiac region is observed, and on palpation a distinct and powerful thud is felt over a considerable area. On percussion, a wide area of dulness is found extending to the left, and below the normal area. It is not so pointed as in simple hypertrophy of the left side, but is more globular, and presents the characters given before as belonging to a combination of hypertrophy and dilatation. This is quite in agreement with what pal-

pation reveals, and percussion and palpation both aid in demonstrating a large, strong, but also widened left ventricle.

But auscultation tells us most of what changes have gone on. The first sound is loud, prolonged, but masked often by a systolic murmur caused by the rush of blood over the roughened aortic orifice. This murmur may be heard up the aorta. There may, too, be heard a soft regurgitant sound at or behind the left nipple, but soon lost. It may be distinguished from the aortic murmur and localised. But the characteristic murmur of aortic regurgitation takes the place of the aortic second sound. Its time singles it out; for the pulmonary valves never almost become insufficient. The pulmonary second sound may be found at its point of maximum intensity, but only with care, for the murmur produced by the backward rush of blood through the aortic orifice is clear and distinct. This murmur is most clearly heard at the base of the heart, and where the current takes it with it to the apex. Not unfrequently it is very distinct at the base of the ensiform cartilage. Wherever heard, it is always true to its time of being heard during the second sound. It is truly diastolic. When combined with obstruction, which is very common, there is a very distinct "see-saw," or rather "saw-see" sound heard over a large area. It may be heard so clearly at the mitral area as to give the impression that it must be produced here, until examination over its more proper area reveals it to be even louder there. The vibrations, too, which to the ear are sounds and a murmur, are apparent to the finger when placed over the base of the heart. The ear when applied, with or without the stethoscope, becomes conscious of the powerful heart stroke which accompanies this condition.

These signs in the heart are aided by signs in the arteries. The aorta, dilated, atheromatous, and roughened, can often be felt at the sternal notch, especially when the patient holds his head forward, and the vibrations can be felt with the finger. The rapid and excessive distension of the arterial system can be felt in the anterior tibial, where the Germans always feel for it. All parts of the arterial system

are affected, down to the smallest artery, as has been stated before. This arterial distension may be felt as a distinct jar over the whole body, and Sir Thomas Watson relates a case of a man whose wife would no longer take his arm when walking out with him, in consequence of the unpleasant blow-like sensation produced by the arterial pulsation.

The symptoms are not peculiar for a long time, and a man may continue to labour for some time after the establishment of distinct aortic regurgitation. But he sooner or later, and usually sooner, has to relinquish his employment; and in the iron districts there are numerous young men so incapacitated. He is anxious, nervous, terribly conscious of the working of that heart. The carotids pulsate very visibly, and are characteristic when seen on each side of an anxious, wistful, and somewhat frightened looking face. A little later he is in bed, propped up, breathing with difficulty, his irregular pulse telling of fatty degeneration gradually enfeebling his heart. Often a process of skin-degeneration, not uncommonly quite fatty, and unctuous to the feel and visible to the eye, is found along with this form of heart disease. The degenerate atheromatous temporal artery may be observed plainly jerking. The appetite fails, a sort of semi-unconsciousness grows over him, and soon the last change follows.

The *prognosis* of aortic regurgitation is not very difficult, unfortunately. It is a form of heart disease which tends to prove fatal very rapidly. The hypertrophy becomes excessive to resist further dilatation from the increased pressure of the distending force; the large and powerful chamber in its turn induces changes quickly in the arterial system, while the law of Mauriac ensures speedy degeneration. The patient in the meantime is liable to genuine apoplexy, though this does not occur very frequently, and he is also very liable to be carried off by intercurrent disease. The prognosis is much affected by the amount of obstruction combined with it; and in the very common cases of this combination the more it partakes of the character of obstruction, and the less of that of pure insufficiency, the better for the patient, and the longer is he likely to live. With care in some cases life may be

prolonged for a considerable time, but only when combined with inaction.

This brings us very naturally to the question of treatment. And though it has seemed best, for divers reasons, to aggregate the treatment in one chapter, as it can only be regarded as built on the same plan in every form of heart failure, still there are some points of special moment in the treatment of aortic regurgitation. The first is to at once insist on the abandonment of all severe labour, and the adoption of some light and more constant occupation. As this disease is so often found in working men, this is a painful point to press steadily and urgently. But it must be done, and the reasons given for it. By reducing the calls on the heart, we can to some extent preserve it; permit labour as long as it can be sustained, and you anticipate that epoch. To attempt to lower that heart's action would be to at once invite dilatation to proceed. The force of the ventricular action, and the increase in the amount or bulk of blood discharged into the arteries at each systole, are rapidly and inevitably ruining those arteries; but it is unavoidable, and is the lesser evil of the two, for it is when that force is beginning to fail that we see the beginning of the end. The recognition of the inroad of this disease in its earliest stages is of the utmost importance, and its etiology is of great moment as giving us data as to when we may suspect its approach, and then we might take measures to delay its advent. After its establishment, each case will present indications of its own, which will be patent enough, and must be treated accordingly; but all treatment is unsatisfactory. At times some relief may be afforded, but usually and generally the treatment is merely palliative, and the profession unite in regarding aortic regurgitation as one of the most serious and, withal, intractable forms of heart disease. The treatment must be conducted according to the rules laid down in the chapter on Treatment, with such modifications as the good sense and intelligence of the reader deem necessary.

Mitral Disease.—In regarding the pathology of disease of the mitral valve, it would serve no useful end, and lead merely to confusion, to separate insufficiency and stenosis. The mitral

valve has already been shown to be involved in some cases of aortic regurgitation. It is often primarily affected, and especially in endocarditis associated with acute rheumatism. Here the destructive process often affects the chordæ tendineæ, till the valve becomes attached to the musculi papillaries themselves; the process commonly creeps along the edges of the valves, and gradually draws the two valves together, until the mitral valve is a rigid cone protruding into the left ventricle, with a narrow, slit-like orifice, commonly called a "button-hole" mitral. This cone is attached immediately to the musculi papillaries, while the endarteriitis creeps over their surfaces in time and distorts them. At other times the process confines itself to gradually thickening and contracting the valve itself, and especially the one which lies near the aorta. Here insufficiency is produced, and not stenosis. In other cases, but perhaps less seldom, the valve, more particularly connected with the outer wall of the ventricle, becomes diseased, and either contracted, or allowed to wash backwards on the systole, from ulceration of the chordæ tendineæ, &c. The various forms of mitral disease are common, both insufficiency and stenosis, and that midway form combining both. The mitral valve, too, is especially affected in those cases of heart disease occurring in young persons, and connected with acute rheumatism; while, on the contrary, aortic disease is rather connected with general atheroma of the arteries in older or elderly people. In mitral disease we come to a series of backward actions, which are not associated with simple aortic disease, but which invariably manifest themselves when the mitral valve is affected, whether primarily or secondarily.

The changes involved by mitral disease affect, first, the vessels of the pulmonic circulation and their terminations at each end. Thus the left auricle is enlarged and thickened, and where there is stenosis, not unfrequently rigid, not collapsing when cut, and with its lining membrane white, opaque, and atheromatous. The pressure to which it is subjected alters it materially, and in time it may undergo degeneration. The vessels behind are firstly distended and thickened, and this extends to the pulmonary artery, which

may become as large and thick as the aorta. This is accompanied by a dilated and thickened right ventricle which forces blood into the pulmonic system under increased pressure. This is most readily detected by the increased sound produced at the semilunar valves. Skoda first pointed out the importance of this accentuation of the pulmonary second sound in mitral disease. This increased tension in the pulmonary circulation leads in time to changes in the thickened vessels. They become rigid and atheromatous, and even in time the subject of fatty degeneration. But this condition of congestion of the pulmonic system leads to many secondary changes in the lungs and air tubes. The nutrition of the lungs is not altogether derived from the bronchial arteries, but is largely supplied by the pulmonary circulation, and a plug in a pulmonary vessel will lead to necrosis of that part of the lung to which it is distributed. So we find increase of connective tissue, leading to atrophy of the lungs and deposits of pigment, especially in young people. We find rupture into the substance of the lung and true pulmonary apoplexy, the clots being round and firm, and not unlike a black bagatelle ball. In older persons, where there is often allied kidney disease and the blood is impure, we have often a deteriorated lung tissue, friable and liable to the formation of emphysema in the repeated paroxysms of dyspnœa which occur. The establishment of emphysema adds to the difficulty of breathing already existing.

The congestion, too, is felt in the bronchial lining membrane and an increased flow of bronchial mucus is usually present, and is called chronic bronchitis, or if profuse, bronchorrhœa. This bronchial flow adds to the patient's distress and often prevents refreshing sleep. Whether it arises from congestion of the pulmonary vessels or true bronchial veins is not of much importance. This pressure on the vessels may lead to passive hæmorrhage, which is often useful as giving relief. As we cannot always discriminate betwixt this hæmorrhage and flow from a rupture in an atheromatous pulmonic vessel, our prognosis must be guarded. There is often œdema of the lungs, especially in the latter stages, and, finally, pleuritic effusion, and, along with the latter,

effusion into the pericardial sac. What changes take place behind the tricuspid must be considered when tricuspid lesion is under consideration; at present we are dealing with the immediate consequences of mitral disease itself. The symptoms produced by these lung changes, &c., are what might be expected, difficulty in breathing, especially on slight exertion, cough, expectoration, and, not uncommonly, hæmoptysis. There are some points of peculiarity about the cough of pulmonic congestion which need pointing out. This cough is commonly found along with heart disease, both in old and young, but is never absent from young victims to mitral disease. It is a dry, imperfect kind of cough, and much resembles a nervous cough, and is quite distinct from the cough which raises phlegm. It is somewhat difficult to describe, but is not at all difficult to recognize after being once observed. It is often a source of considerable annoyance, and tempts the patient to try cough medicines, which often do great harm, especially as the indications seem to point to paregoric, or other medicaments containing opium. It is only to be treated by measures which affect the condition on which it depends.

At the same time, caution must be exercised in discriminating betwixt the increased flow of bronchial mucus from a passing condition of excessive pulmonic engorgement and an attack of acute bronchitis. This latter is almost invariably fatal when it occurs in a person with chronic mitral disease, the former often threatens life gravely, but is commonly enough recovered from. Mere flow from congestion lacks the signs and symptoms connected with the income of an inflammatory attack. These symptoms are all common to mitral disease, whether insufficiency or stenosis. But it is of great matter to form a correct diagnosis, especially as regards prognosis.

Mitral Obstruction.—We will take first mitral obstruction according to our order. Mitral obstruction, depending as it does on obstruction existing betwixt the left auricle and ventricle, has a murmur, often a distinct one, found immediately before the ventricular systole. It is pre-systolic, and not diastolic as described in older works. This makes it

peculiar and special, for though I have read two cases, one by Bamberger and the other by W. T. Gairdner, which were deemed obstructed tricuspid, both turned out to be caused by tumours pressing on the right auricle. Tricuspid obstruction is as yet unknown.*

We have, then, a murmur heard at the tricuspid point of maximum intensity in the fourth intercostal space about an inch in front of the nipple, which is pre-systolic. It is accompanied by chest symptoms, by accentuation of the pulmonary second sound, and right side enlargement. There is no increase in the size of the left ventricle, and the pulse is small, compressible, but regular—quite regular. It is commonly found in the young after rheumatic fever, and its prognosis is bad, in consequence of the gradual progress of the pathological changes on which it depends, and the impossibility of compensatory changes being long effective.

In *mitral regurgitation* we have some points of important difference from mitral stenosis. The sound or murmur is heard not only from this point in the fourth intercostal space in front of the nipple, but is heard even sometimes away behind the nipple as well, and even in some cases reaches its maximum intensity about an inch, or even more, behind the left nipple. In some cases the murmur is confined to these areas, in other cases it can be heard all over the back. The sound is systolic, and may therefore be confounded with aortic obstruction, the points of distinction having been already given. It is invariably accompanied by dilatation and more or less hypertrophy in the left ventricle, as well as the changes in the right side common to all mitral disease. The changes in the left ventricle, as Niemeyer shows, are due to the distension of the flaccid chamber by blood driven in under unusual pressure. At each ventricular systole so much blood is driven backwards through the incompetent mitral; this bulk of blood is added to that already in the distended pulmonic system, driven in by a thickened right heart. Consequently, when the blood again rushes into the flaccid chamber, it does so under greater pressure, and consequently we have dilatation with more or less hypertrophy.

* Except when congenital.

Another point about mitral regurgitation is its peculiar pulse. This pulse will itself often form a sufficient ground for a diagnosis to a competent finger. It is obvious that whatever bulk of blood passes backward on each systole, so much less will pass forward into the aorta and arterial system. Why this should be more at one time than another, it is impossible to say. This difference is felt in the radial pulse, and constitutes irregularity in volume. That is its peculiarity, its variableness in volume. This is quite distinct from irregularity in time, even when they are found together, as is common enough in the later stages of the disease. Mitral regurgitation is rather a disease of advanced life, and often permits of a good prognosis as to time.

Mitral Obstruction.	*Mitral Regurgitation.*
Murmur limited, and pre-systolic.	Murmur not limited, often heard backwards, and systolic.
Never accompanied by changes in left ventricle.	Always accompanied by more or less change in left ventricle.
Pulse small and regular in bulk and time.	Pulse irregular in volume, varying much, and often in bulk, and in the latter stages in time.
Found commonly in young subjects.	Found in young subjects, but equally in old persons. Often found in connection with aortic disease.
Found alone as regards left side of heart.	
Prognosis as to time very bad.	Prognosis as to time often very fair.

Points common to each.

Frequency of accompanying tricuspid disease.	Frequency of tricuspid disease.
Changes in right heart invariable.	Changes in right heart invariable.
Accentuation of second pulmonary sound.	Accentuation of second pulmonary sound.
Cough and other chest symptoms.	Cough and other chest symptoms.
Prognosis as to life bad.	Prognosis as to life bad.

This chart points out the points of resemblance and of difference. Often, however, are found cases presenting more or less of points special to each. These mixed cases, as in aortic disease, must be classed according to the one they resemble most, with the other added as a qualification. The prognosis, too, will go with the form which it most resembles. As the difference in prognosis has been inserted in the chart, it may be desirable to give the grounds for it.

Prognosis.—The prognosis in mitral disease is founded on the pathological changes. Thus in stenosis the process of

matting the valves and chordæ tendineæ together, admits of no compensation; and the obstruction thus offered soon ruins all parts behind the lesion. The progress of the case thus is more rapid. But in mitral regurgitation the lesion is often slow in its growth and confined to the valve on the aortic side. As this becomes shrivelled and smaller, the general dilatation of the ventricle has affected the outside wall and its valve, lengthening and elongating the musculi papillaries and chordæ tendineæ. Thus the valve of the yielding wall is still stretching across a great part of the orifice, while the pressure of the blood behind aids somewhat in preventing regurgitation. Taking the whole circumstances together, mitral stenosis is a disease whose tendency is to pass rapidly from bad to worse, and is soon fatal, whereas mitral regurgitation has more tendency to remain in *statu quo*, advances slowly, and admits of some, though imperfect, compensation.

Treatment.—There are no particular points to be attended to in the treatment of mitral lesions. The treatment must be conducted on the general principles. Digitalis can give only temporary and fleeting relief in mitral stenosis. In mitral regurgitation, it often strengthens the right heart and enables the left ventricle to resist the increased distension, and is often of great and lasting service.

Diseases of the Right Side of the Heart.—We now come to diseases of the right side of the heart, when we have a totally new series of phenomena added to those already described: when the disease of the right side is secondary to disease of the left side, that is in the great majority of instances, and almost invariably as regards changes in the valves. Even as secondary affections, diseases of the pulmonary orifice do not furnish us with much to describe as regards pathology, while the diagnosis is most difficult.

Diseases of the Pulmonary Orifice.—If diseases of the pulmonary orifice were not so infrequent, we might perhaps know something more about them. As it is, we know that the pulmonary orifice may become affected secondarily to disease of the aortic orifice, that this may entail either stenosis or insufficiency, or that the pulmonary valves may

become affected by the extreme tension in the pulmonic circulation in disease of the mitral valve.

Still it is rare to find the pulmonary valves affected even in great obstruction to the pulmonic flow, and a small festoon-looking deposit of atheroma in the valves is not even usual. The pulmonic orifice is occasionally narrowed by congenital stenosis at the conus arteriosus, but this belongs to another chapter.

As to the diagnosis of pulmonary lesions, it is arrived at by exclusion chiefly. The positive points being sensible superficiality in the sounds, absence of all sound at the back, and entire absence of disease of the left side of the heart. More than this it is scarcely safe to say, except that the pulse at the wrist would not have the peculiarities imprinted on it which would be found were the disease at the aorta. As to the time at which they would be heard, &c. Pulmonary stenosis has a feeble sound discernible over the third left costo-sternal articulation, limited in its area, not heard in maximum intensity at the aortic area, the second right costo-sternal articulation, and, of chief moment, not communicated along the aorta and great vessels. There is evidence of right side hypertrophy and absence of left side hypertrophy.

So, too, in the almost unknown cases of pulmonary regurgitation, the diagnosis was established by the foregoing rules for exclusion, and by the absence of that pulse so peculiar to aortic regurgitation. But the establishment of a diagnosis of pulmonary disease is a difficult task for the most accomplished auscultator, and can scarcely be expected from the majority of those for whom these pages are specially intended. Not that they should not try, but that they must not feel chagrined if they are not quite successful in avoiding error.

The prognosis of pulmonary disease is essentially bad, and its addition to left side disease hastens the end of the case. Also when occurring primarily it may be fairly presumed that its effect on the right ventricle is to produce in it and in the tricuspid valve, those changes which close the scene of left side disease, when the right side has become implicated.

The comparative frequency of disease in the right and left sides of the heart in intra-uterine life is reversed: then disease of the right side is much more frequent, and disease of the left side almost unknown.

Tricuspid Disease.—The extreme rarity of stenosis of the tricuspid valve, if indeed it ever exist, has not enabled us to give any positively observed data for it; but it is not difficult to see that a pre-systolic murmur at the base of the ensiform cartilage would be its chief sign, and that general venous congestion would follow it, and that too to a marked extent. On these data a diagnosis has been formed, but in the only two cases which I have seen reported, in each case a tumour pressing on the auricle has been found to have occasioned the murmur and the symptoms. Under these circumstances there is nothing to say as to how a diagnosis should be established. This is of less moment, as it is extremely unlikely that the reader will ever be called upon to make one.

But regurgitation through the tricuspid is not at all an uncommon form of valvular lesion, especially as a secondary lesion; as a primary lesion it is somewhat uncommon. Dr. Peyton Blakiston has paid great attention to this lesion, and has demonstrated that it accompanies left side disease much more commonly than was previously surmised. It is often found in connection with disease of the mitral valve. Many years ago, in a very clever article, Dr. T. King tried to establish that there existed what he called "the safety valve action of the tricuspid," an idea which is still quoted in many works on heart disease and physiology. His experiments were, however, imperfect, and his conclusions hastily drawn, for so far from tricuspid regurgitation having any protective action, it really inaugurates the most serious secondary changes. But the experiments of King need much careful investigation to demonstrate their inaccuracy, and for those who care to investigate the question, they may refer to a paper by the writer, in which this is examined in connection with the hearts of the diving mammals, &c.*

When the tricuspid becomes imperfect, it may possibly arise from sudden engorgement of the right ventricle, but

* "Edin. Med. Journ.," Dec., 1870.

this is only as a temporary condition, and it is only possible. Nor does this condition when more permanent lead to persistent tricuspid regurgitation, for, as Rokitansky shows, this dilatation of a chamber is accompanied by dilatation of the valve, and chordæ tendineæ and musculi papillaries, so that dilatation does not lead to insufficiency of the auriculo-ventricular valves as long as that valve apparatus is itself not the subject of disease. The commonest pathological condition which leads to tricuspid insufficiency is the same as in the mitral, viz., shrivelling and distortion of the small valve of the fixed wall, with its cords. This sometimes proceeds until the tip of the forefinger can scarcely be inserted betwixt the valve and its wall. Similar changes may affect the other portion of the valvular apparatus, but less commonly. The right auricle is not nearly so liable as the left to suffer from endarteriitis deformans, and does not become so altered by distension. It may hypertrophy, true, but it is more essentially venous, and suffers rather passive stretching. Under this stretching it sometimes becomes extremely thin and transparent, and when held up to the light is seen as a nearly transparent fibro-serous membrane with muscular fibres at intervals.

The changes in the right auriculo-ventricular valve have often been attributed to a sort of sympathy of one side with another—a fact about some ailments; but in this case we have the more direct explanation that until the mitral is affected no stress is thrown on the right side of the heart. This stress leads to dilatation and hypertrophy of the right ventricle, and from that more stress on the valves and their attachments. This is more probable, and is rendered more so that one of the best marked cases of tricuspid regurgitation the writer ever saw (so far as a diagnosis can be accepted that is not corroborated by *post mortem*) occurred immediately after an attack of pneumonia in an old man of 84. The symptoms were all marked; dropsy set in while he was still walking about, and in two months all was over. There may be something in the sympathy betwixt the two auriculo-ventricular valves, but we must be guarded about it when another much more obvious cause is in operation.

We will follow now the secondary consequences of tricuspid insufficiency. When this occurs it is obvious that at each ventricular contraction a backward rush of blood will over-distend the veins behind. These veins are to some slight extent muscular at their terminations, and as such contractile. This and the contracted condition of the auricle will to some extent impede the backward flow of blood; but its great stress falls on the elasticity of the veins, which is already considerably involved by their engorgement. The engorgement of the venæ cavæ leads to general venous stagnation. The coronary circulation is pretty well protected against venous stagnation by the separate outfall of its veins into the right auricle. The stagnation in the venous system is not equable in consequence of the venous valves.

Thus the internal jugular prevents direct regurgitation into the circulation within the encephalon, while the countenance is early engorged. These venous valves, too, prevent regurgitation into the arms and lower limbs. The great early stress is thus thrown on the portal circulation, which, by its external openings can most quickly relieve itself by natural discharges. This fortunately enables the system to continue its existence a little longer, even under a very advanced condition of disease. We have, as related in a previous chapter, congestion of the liver, and in the young, rapid development of connective tissue and the formation of liver cirrhosis. So, too, in the old; though in the old we often find a condition of vascular engorgement of the liver, with notable increase in bulk. The Germans state that this regurgitation into the veins of the liver makes a "liver pulsation" isochronous with the heart's contractions. But to this we will return. Also congestion of the bile ducts, alterations in the bile itself, &c. Catarrh of the stomach and intestinal canal are very common, and a source of trouble to the patient; hæmorrhoids, &c.

The spleen is enlarged or cirrhotic. The kidneys begin to suffer from this venous congestion, and the urine is scanty, laden with urates and albuminous, whatever may have been its condition at an earlier period. Increase in its interstitial tissue is marked, and often may be seen marks of old standing

mischief amidst the new formation. There is in women mennorrhagia, metorrhagia, and leucorrhœa, and in men prostatic enlargement and hæmatocele (Oppolzer). There is effusion into the peritoneal cavity generally. But sooner or later the distension of the veins leads to insufficiency of the venous valves themselves, and then we have œdema of the feet, spreading upwards into the scrotum, &c. In spite of the authority of Rutherford Haldane, Oppolzer, and others, it must be admitted that we often have dropsy, which passes away, and does not return for long, which could scarcely be the case if the venous valves were become insufficient, or the tricuspid affected. Still no doubt when the venous valves are become insufficient, persistent dropsy follows and remains. The valves of the internal jugular become affected, and drowsiness, harassed by horrible dreams, sets in, gradually increasing to coma. Again, there may be a great deal of venous congestion within the encephalon long ere the venous valves become insufficient. The venous flow can only be somewhat limited through those valves forward when opposed by great congestion in the venæ cavæ, and an observable amount of congestion of the contents of the encephalon may and does commonly occur long before there is any decided effusion into the ventricles, or increase in bulk in the cerebro-spinal fluid, &c., which follow quickly when the valves of the internal jugular become affected. This cerebral congestion is first apparent in the mental manifestations, in disagreeable dreams when asleep, in capriæ, petulance, irritability, erroneous calculations, rash speculations, coldness, or suspiciousness of old friends, tendency to form new connections in opposition to old ones, and, indeed, a thousand alterations, which are very trying to the friends and the medical man, while they must also be grave trouble to the patient himself.

The *diagnosis* of change in the right ventricle, &c., when following left side disease, is not at all peculiarly difficult. The changes in the right side of the heart in primary right disease are not so well marked, as the disease rarely remains long enough to develop them well. In the wake of left side disease, and in addition to the changes previously inaugurated,

we get a new series of changes, to inspection, palpation, percussion, and auscultation. On inspection we find a diffused heaving, extending from the right apex in a curve towards the epigastrium, and even to the right second intercostal space; indeed a distended and hypertrophied right auricle may often be seen playing at that point.

Palpation assures us of this, and distinguishes the superficiality of the changes as compared to left side disease. The distended right ventricle lies more upon the diaphragm than in health, and the heart itself lies more horizontally across the chest than under other circumstances. The left apex beat is often undistinguishable, unless there be left side hypertrophy, and then it may be felt beating rather to the left of the normal left apex. This position of the right ventricle communicates its impulse to the liver, and especially the left lobe; this is the great cause of the "liver pulsation," though it is possible that the venous regurgitation into the liver may aid in producing it. We can also feel the right auricle in action in the right second intercostal space.

These two aids will materially assist us in percussing correctly the outlines of the dilated and hypertrophied right heart, and to distinguish betwixt the sounds produced by the various organs, for to map out the right side of the heart is more difficult, and contains more elements of error than does mapping out the left side by percussion. Still percussion, with its accompanying sensations of resistance, will often be of aid to us in examining the right side of the heart, and more so as regards the right base and the right auricle, the latter often enabling us to describe its boundaries by percussion.

Auscultation will tell us more of the changes which have gone on. In the first place is the important accentuation of the second sound at the pulmonary valves. Then there is the loud and strong sound of the increased muscular contraction, and so long as the tricuspid is not much affected, a sharp sound from the auriculo-ventricular valves, driven together with increased force. But when the tricuspid has become affected, this sound is no longer heard, and in its place is a soft blowing sound heard over a most limited space

of area. Just at the point of the ensiform cartilage, and nowhere else, is this sound heard. It is unique, and not likely to be confounded with mitral regurgitation or aortic regurgitation. True it is, that if in a case of supposed tricuspid regurgitation, a stethoscope be applied over the tricuspid area, a murmur faint and low may be heard, but it can be traced away, and located in another quarter. Often it can be traced to the nipple, or even behind it, in mitral regurgitation. Sometimes, too, an aortic regurgitant murmur will be heard with great clearness in the tricuspid area, but it can also be traced elsewhere and fixed. This murmur is significant when heard, but it is often wanting, and even after being distinctly audible for a while, be not longer so as the patient grows nearer his end. This may be that the regurgitation is not marked enough, or sufficiently powerful to any longer give rise to an audible murmur. In the same way, as needs pointing out more especially, when tricuspid regurgitation becomes marked, the murmurs of the left side become inaudible. In two well-marked cases at least the writer saw this, where extensive aortic obstruction co-existed with a "button-hole" mitral; in one case no murmur could be heard in the left side, and in the other only a faint one over the pulmonary area. The effect of tricuspid regurgitation in masking left side lesions needs more observation. It seems probable that in both these two cases the tricuspid regurgitation permitted so much backward flow of blood, that the blood stream in the left heart was unequal to eliciting audible evidence of obstruction offered to its flow.

We often find, too, in auscultation of the right heart irregularity, which is not so readily discernible in the radial pulse, especially in mitral stenosis. Here the right heart is in great distress often, when no difficulty is offered to the flow of blood from the left ventricle. This irregularity, often combined with palpitation, in the right heart, as almost confined to it solely, is not uncommon, and specially in the attacks of cardiac dyspnœa, to which this condition is liable. In fact, very often the objective symptoms are really connected with the condition of the right heart, though when becoming marked they are communicated to the left heart by

the muscular fibres common to each, and by it to the radial pulse. Cardiac excitement is often really right side, and in these conditions there is no difficulty in so diagnosing it. This condition of the heart and tricuspid regurgitation are also manifested by "jugular pulsation." This when marked may be regarded as pathognomonic of tricuspid insufficiency, as the valves in the narrow portion behind the sterno-cleido-mastoid only become insufficient from such dilatation as is produced by tricuspid regurgitation only. A finger properly applied will arrest it. This distinguishes it from carotid visible pulsation, which often occurs in aortic regurgitation. But the absence of jugular pulsation does not establish the fact of the non-existence of tricuspid regurgitation in its earlier stages; its presence proves that marked tricuspid regurgitation is fully established, and that the regurgitation has become sufficient to dilate the veins until the venous valves become visibly insufficient.

Prognosis.—The prognosis of tricuspid regurgitation is a very important part of it. So important, indeed, is tricuspid insufficiency in its effect upon the prognosis that the prognosis may truthfully be said often to hang upon the resistance offered by the tricuspid.

So long as the tricuspid remains sufficient, there is an element of hope as regards duration of life in the case. With one valve left sound behind a muscular chamber, something may be done, and the right ventricle may yet be acted on, and some compensation maintained. But let the last valve become insufficient, and the aspect of the case changes at once. There is nothing now left but the feeble barrier of the resistance of the veins already distended, and now to be subjected to the direct distending force of the backward rush of blood propelled by a dilated and hypertrophied ventricle, a force veins were never intended or constructed to withstand. To this new distending force the veins rapidly yield, the last changes commence, and the case is quickly hurried to a termination. So when tricuspid disease is primary, the case soon runs its course. For that regurgitation under ventricular impulse is ruinous to the veins, and the case

commences about the point when an ordinary case of heart disease is putting on its last and worst aspect.

Treatment.—It is obvious from the pathology of tricuspid regurgitation, that any attempt to act on the heart could hold out no prospect of success; it could only increase the force of the right ventricle in destroying the veins behind. All treatment then, must naturally belong to the last division of treatment of heart disease, viz., the treatment of sequelæ. Drastic cathartics are the most effectual means at our disposal, and five or six copious fluid motions relieve the venous engorgement, including the cerebral congestion, and for a brief period relief is experienced. No diuretics can be expected to be of any avail here, for with that leaking tricuspid it would be futile to attempt to increase the arterial pressure. Incisions, or needle-pricking, may be resorted to, but they contain no hope of any real relief, and make the patient's bed, &c., very uncomfortable.

CHAPTER VIII.

DISEASES OF THE MUSCULAR WALLS OF THE HEART: MYOCARDITIS—FATTY DEGENERATION—SYMPTOMS, PATHOLOGY, &C.—FATTY INFILTRATION—CONNECTIVE TISSUE HYPERTROPHY—ATROPHY—AMYLOID DEGENERATION—SYPHILITIC GUMMATA—TUBERCLE—CANCER—POLYPI.

IN describing the diseases of the muscular walls of the heart great hesitation will often occur in consequence of the difficulty of the subject, and the acknowledged imperfection of our observations. A great deal has, undoubtedly, been cleared up, but much yet remains to be learnt. Not only are the pathological changes themselves, both gross and histological, still somewhat obscure, but of the causation in many cases we know absolutely nothing. We can understand diseases of tissue, chiefly degenerative, arising from such obvious causes as embolism or ossification of the nutrient artery; or that the heart-wall, in common with other muscular structures, is the seat of syphilitic gummata, cancer, or tubercle. But as to changes commencing in a low inflammatory process we know, causatively, absolutely nothing. To what extent these changes are connected with derangement of the complex nervous supply of the heart, as to whether there is disturbance in the vaso-motor nerves, the nerves commonly called trophic, or in both, or even neither, we cannot yet even speculate. The difficulty of the subject, the absence of the necessary preliminary knowledge, both of nerve-inflammation and of inflammation of muscles, sufficiently explain the absence of observations yet, and for some time to come, in all probability. It must be confessed that there is much that we could much like to know, and would willingly and eagerly learn, but it is not yet possible, and we must rest, not satisfied, but patient, for the present with what is at our disposal. It will then be the writer's aim to

give an account, as clear as is within his power, of the present state of medical knowledge, perhaps sometimes rather medical opinion, on the various changes which go on in the muscular walls of the heart, commencing with changes connected with inflammatory action, and then describing those changes, which can scarcely be called inflammatory in the ordinary acceptation of the word. Inflammation of the muscular structure, or myocarditis, will come first, and, after it, the slower or later changes will follow, as fatty degeneration, false hypertrophy, atrophy, and the chronic inflammations of special character as syphilitic gummata, tubercle, &c.

Acute Myocarditis.—Rindfleisch commences his account of myocarditis by assuring us that it is the darkest chapter in all pathology, with which scarcely comforting assurance it is impossible to differ. Inflammation of all muscular structure is very imperfectly understood, and is not so frequent as inflammation in structures where we have a secreting function, which is a great cause of their derangement. Myocarditis when acute is usually more or less general, and is found in pyrexiæ, especially when sœptic in character, and along with endocarditis and pericarditis. It commences, according to Virchow, either in the muscular bundles themselves (parenchymatous myocarditis) or in the intermuscular areolar tissue (interstitial myocarditis). This sometimes runs into pus, and then it is denominated purulent myocarditis. The structure of the heart is at first infected and of a dark red colour, and later becomes rather a greyish yellow, or greyish red, with accumulations of pus in minute masses. Under the microscope the primitive bundles of muscular fibre become increased in bulk and swollen with fluid, and the cross markings become indistinct. To the touch the heart walls feel soft, boggy, and easily torn, and the structure is easily broken up into irregular fragments. The accumulations of pus often run together and form an irregular abscess, which may open through the endocardium into the circulation.

This form of myocarditis is commonly found in pyœmia, where the name metastatic is often applied to it, along with endo-metritis or puerperal fever, typhus, scarlatina, and

Oppolzer states not rarely with cholera. It has been noticed as a common cause of death in relapsing fever, where patients not uncommonly die suddenly in the convalescence from some exertion which is too much for the weakened heart. In what myocarditis differs from the wasting which goes on in muscular structure ordinarily when any heightened temperature is maintained for some time continuously it is impossible to say. It is possible that it may be found rather a difference of degree than of kind. That there is a great debility in the heart in many febrile cases we know quite well, and in the dead-house impaired structure of the heart walls is rarely absent in puerperal endo-metritis; but at this point the disease assumes an unquestionably fatal character, and how far it may proceed compatibly with recovery it is impossible to say.

Symptoms.—There are no positive symptoms of myocarditis, and Oppolzer states that it is not easy to find a disease with so dark a combination of symptoms (symptomencomplex) as in myocarditis. Like all affections of the heart walls there is not a prominence of objective symptoms, and palpitation is not usual, while the subjective symptoms are those common to all cardiac failure. A fast, feeble, compressible, and even uncountable pulse is perhaps the most reliable of all symptoms. Whenever there is acute debility in the heart the contractions become increased in frequency and diminished in force in proportion to the debility, whether due to myocarditis or not. There is often, too, pain in the region of the heart, and it is to the myocarditis which accompanies endocarditis and pericarditis that the pain is stated to be due which so often accompanies these affections, and yet is not necessarily present in them. But in acute myocarditis pain is commonly absent; its presence is of some value, but its absence proves nothing. There is not unfrequently pain in the articulations; but then, again, acute myocarditis is so rarely, if ever, found alone, and without some general affection, that it is impossible to say what general symptoms belong to it, and what to the systemic affection. Probably extreme rapidity of the pulse, with great weakness in it, is the most reliable sign of myocarditis when acute and diffuse.

It is said that the blood is dark and of a violet colour, but that is a post-mortem appearance, which is of no use in life, and even if hæmorrhage did occur, the difficulty of the accompanying systemic affections would effectually prevent any diagnostic value being attached to any peculiar appearance in the blood.

Prognosis and Terminations.—It is to be feared that about a disease whose existence is rather a matter of speculative probability, founded on the revelations of the dead-house, than demonstrated by positive evidence of its presence, it is difficult to express any but the most guarded opinion. Much would depend on the nature of the general complaint, and a very difficult prognosis is probably warranted in relapsing fever with evidences of great cardiac debility, where the presence of acute myocarditis is somewhat more than a mere probability, than in similar circulatory condition in pyœmia or puerperal fever. It must be admitted that in the present state of our knowledge our prognosis must be formed rather on the general condition and the fatality of the general malady than on any circulatory signs or symptoms that might in each case be deemed to be due to myocarditis. We all know well enough that any high mounting of the pulse, with corresponding feebleness of it is a bad prognostic sign in all acute diseases, and especially if it in time be accompanied by irregularity. How far a somewhat excited action of the heart, giving a fictitious appearance of strength not found in the pulse, would enable us to ascertain a greater probability of myocarditis, as an awkward complication in any acute affection, is as yet unascertained; it is merely probable. As to the terminations of acute general myocarditis, it must be admitted that there is some reason for supposing that it is not rarely followed by recovery. Those cases of relapsing fever, &c., where there are the same suspicious appearances in the circulation, which recover, and these are the majority, can differ but little, if at all, from those where some sudden effort or exertion has been followed by a fatal result. There are grounds for thinking that it is frequently followed by recovery, as well as the corroborative evidence furnished by scars (the scars of Dittrich) which are

commonly found on hearts in the dead-house, and which are evidently the remains of some former affection, while at the same time the condition in which hearts are also found, on section, after acute pyretic affections, leaves no doubt in the mind that in many cases the affection of the heart must have been a factor, if not the chief one, in producing the fatal result. But as to when, or how, we are to determine that the affection still continues at a point compatible with recovery, and when that point is irrevocably passed, we are yet in a darkness from which we can only hope some day to emerge.

Treatment.—This can only be general, and such as we should apply in any similar condition. Antiphlogistics, so called, which are supposed to owe their efficacy to their lowering effect upon the heart, are obviously out of place here. Agents which will keep it itself going are indicated, and among these in acute affections of the heart and its serous membranes; the Germans place digitalis first. Stimulants and food of an easily digestible character are indicated, but each case must of necessity rest for its treatment on its own peculiarities. One thing is certain, that in the convalescence from all pyrexiæ, where there has been myocarditis, or a strong probability of it, all effort and exertion must be strictly forbidden. Every one with much experience of a fever hospital knows but too well that every now and again convalescent fever patients die suddenly, especially after exercise, and that death is commonly referable to over-taxation of the heart, weakened by myocarditis.

Chronic Myocarditis.—The heart wall is liable to other changes of a more gradual character than the affection described above, but which are still of an inflammatory nature, and these may best be described as chronic myocarditis, as distinguishing them from the acute myocarditis just described. These changes are usually of a more local character, and are not so general or diffuse. The favourite seats are the left ventricle, and, according to Rokitansky, by preference, the apex, the right ventricle, more rarely the right auricle, while the left auricle possesses almost entire immunity. These changes are various, but commence in an inflammatory process in which the heart fibre becomes altered as described

above, but more slowly, and in some instances not to the same extent. Thus a process may go on, in the left ventricle chiefly, which may lead to such softening of the heart fibre as will lead to the formation of cardiac aneurism, that is, bulging outwards in the affected part, while at other times a complete abscess may be formed, by the molecular death of the part supplied by a small vessel closed by an embolon. There are various stages reached which have not yet been sufficiently examined microscopically to demonstrate their pathology thoroughly. A description must then be confined to the naked-eye changes, and, as this local myocarditis is not itself fatal until rupture of the heart walls occur, it is only the more advanced results that come under notice. The earlier stages are not presented to us, or so rarely, that no account of them can yet be constructed. It would appear that this myocarditis is liable to proceed further at some parts than others, as if a point were reached in the process when repair became impossible, while the surrounding tissue becomes perfectly restored. The evidence of this is found in the scars and puckers, so commonly seen in the dead-house, showing where old destruction had gone on, and scattered over the surface of the heart, more or less, and giving to the affected part a wrinkled appearance. This inflammatory process, with its ultimate formation of contractile cicatricial tissue, is sometimes found near the right or left conus arteriosus, and then forms an obstruction to the flow of blood, and is called the true cardiac stenosis of Dittrich.

At other times the inflammatory process produces merely a softening of the affected tissue, which causes it to yield before the distending force of the blood in diastole, and then is formed a bulging, or true cardiac aneurism. This is most commonly found in or near the left apex, but has been found in the septum ventriculorum. It may vary in size from a small aneurism no larger than a bean up to a size equal to the heart itself. The visceral pericardium stretched over it may become almost lost and invisible, while the endocardium lining it may become atheromatous or covered with villous vegetations. There is always great risk of these aneurisms bursting, and more so if the heart, from any cause becomes hypertrophied,

for the more the pressure centripetally is increased in the sound, acting, parts of the heart wall, the greater must be the centrifugal pressure on the parts which are not in action. The formation of partial cardiac aneurism (to distinguish it from general eccentric dilatation, which of old was sometimes called cardiac aneurism) has been studied and described in our own country chiefly by Canton and Wilks.

Sometimes the papillary muscles are especially affected by this chronic inflammatory action, which is degenerative in its character. In these cases there is auriculo-ventricular regurgitation from imperfect closure of the valves in consequence of inability in the affected papillary muscles.

At other times when there has been more acute myocarditis, with here and there the formation of small accumulations of pus, on the recovery of the inflamed portion, the masses of pus become encapsuled in a pyogenic membrane of connective tissue, and are thus rendered no longer a source of danger from rupture. When myocarditis is, however, the result of embolism of one of the smaller branches of the coronary circulation, as occurs after suppuration or destruction in the lungs, the portion of muscular wall thus deprived of its nutrition (for there is no communication betwixt the coronary arteries and thus formation of collateral circulation; Quain) becomes not perhaps so much inflamed as subject to molecular necrosis, and the formation of abscess. This untoward accident may lead to death by perforation of the heart wall directly, or may discharge itself into the circulation, and form cardiac aneurism, which should strictly be called "false," as distinguished from "true" cardiac aneurism, and thus produce a fatal result, from rupture of the sac at some later period.

Symptoms.—The symptoms of partial chronic myocarditis are somewhat obscure, and in the case of small scars probably no special symptoms are found. At other times there are pains in the chest, dyspnœa, impeded circulation, feeble pulse, with increased rapidity and irregular action in the heart itself. There may be palpitation even in the sound portions of the heart. It was thought by Piorry that he could detect a peculiarity in the pain of myocarditis which

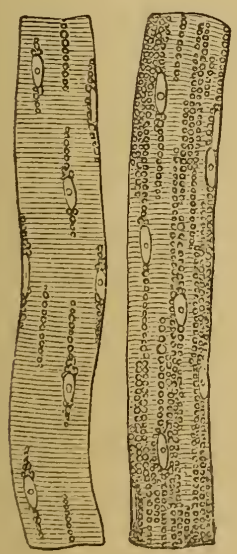
rendered it pathognomonic, and that was its exacerbation on systole. He thought that it was possible for the sufferer to distinguish an increase in the amount of pain on each ventricular contraction. There is an *à priori* objection to this in the rapidity of the pulse, and the fact that some time is required in the transmission of sensations by the nervous system, while impressions rapidly repeated acquire the character of continuousness. No other observer has been able to elicit this peculiarity in the pain of myocarditis, and it is only necessary to allude to this idea of Piorry's to prevent youthful readers from wasting time in attempting to find it. When there is aneurism of the apex of the heart, Skoda has thought it possible to detect an intensified bulging of the corresponding intercostal parietes, and he is entitled to credence from the accuracy of his observations. Where there is the stenosis of Dittrich, a systolic murmur will be heard at the base of the heart; and when the papillary muscles are affected, a systolic murmur at the right or left apex, or rather at the ensiform cartilage, or the left nipple. The contractions of the ventricle are usually somewhat feeble and unrythmical, and occasionally unusually slow; but the diagnostic value of this last symptom has not yet been appraised.

Prognosis and Terminations.—The course of chronic myocarditis is so various that the difficulty of diagnosis is quite equalled both by the difficulties of prognosis and of treatment. The prognosis can only be founded on general symptoms and experience, the cases are too rare to enable any special rules to be formed. When this symptom of Skoda is observed, it will indicate a condition liable to sudden death, only to be met by guarded inactivity, and where an unfavourable ultimate prognosis is warranted. The subject of myocarditis is too shrouded yet to admit of any dogmatising, and the medical man, who can diagnose it, is, in my opinion, quite capable of forming a prognosis and deciding on a line of treatment for himself without any extraneous aid.

At other times no doubt myocarditis is the precursor of fatty degeneration and other chronic conditions of the heart walls of sufficient importance to entitle them to separate consideration.

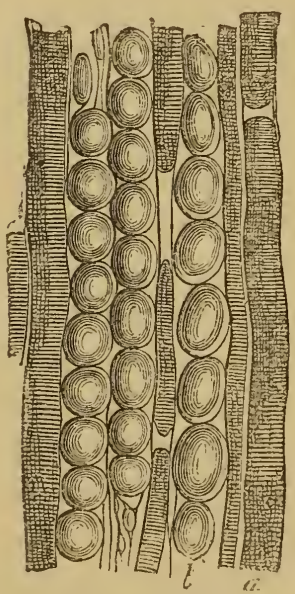
Fatty Degeneration.—From inflammation of the muscular structure of the heart we now come to a less unknown, but still difficult subject, namely, fatty degeneration of the heart. In consequence of confusion in nomenclature, as well as two distinct pathological processes, each fatty in a totally different way, it will be necessary to be somewhat lengthy in order to be explicit on this subject. There are two morbid processes connected with the development of fat in the heart, which were at one time confounded, but which are distinctly different. To establish the best of all distinctions, a broad anatomical one, it may be stated that the one is an affection of the muscular fibre itself, the other an affection of the intermuscular connective tissue. The one is connected with the degeneration of the muscular fibre, allied to the fatty degeneration of a post-parturient uterus; the other is a development of fat in the areolar tissue, a true interstitial growth, which affects the muscular fibre merely by pressure on it. The difference will be most strikingly demonstrated by placing side by side these two plates of the different conditions, taken from Rindfleisch's "Pathological Anatomy."

FIG. VII.



Fatty Degeneration of Heart Fibre, showing more and less advanced stages (after Rindfleisch).

FIG. VIII.



Fatty Infiltration of Heart (from Rindfleisch).

The distinction is clearly apparent; the one is an affection of muscular fibrillæ, and the other of the interstitial connec-

tive tissue. With the second form we are not yet engaged; it will be considered afterwards, along with the other affections of the connective-tissue of the heart.

True fatty degeneration of the heart is a consequence not rarely of parenchymatous myocarditis, especially in the more acute conditions associated with relapsing fever, puerperal fever, &c., and notably phosphorus poisoning (E. Wagner). As such it is properly considered next in order to myocarditis; but it is in its more frequent and chronic condition a degeneration, which Dr. R. Quain has described as the result of a chemical or physical change in the composition of the muscular tissue itself, independent of those processes which we call vital.—(Lumleian Lectures, 1872.)

In order to elucidate this subject, it may not be undesirable to wander, for a brief time, from the exact subject in hand. Fatty degeneration is a process certainly not necessarily connected with life : in fact it is somewhat antagonistic to the idea of vitality, it is a species of molecular death. It is, however, a normal process, as seen in the uterus after delivery, where the mass of muscular fibre, having fulfilled its function, undergoes involution by resolution of its tissue into fat and ammonia. So, too, muscles which are suddenly cut off from their nutrition, as by ligature of an artery, undergo fatty degeneration, until their normal nutrition is restored by the establishment of collateral circulation. In fracture also, where muscles are prevented from use for a time by our surgical appliances, this disuse leads to a passing degeneration. The decay of the formed material of the muscular fibres takes the form of fatty degeneration, when no longer normally removed and replaced by new material. There is a sort of death in the process, that is, the decayed is not replaced by new material. For this same fatty degeneration occurs in muscle when placed in running water, or in a solution of nitric acid and water, very highly diluted. The development of fat, or adipocire, is a gradual process in which the muscle becomes paler, softer, less consistent, and ultimately is found a piece of dirty white fat, all structural formation being lost. The gradual development of fat in the muscular fibre can be watched under the microscope, and

here there can be no question that the fat is the result of retrograde metamorphosis of the muscular fibre, and not any infiltration of fat from without. The complex albuminous, or protein compounds, are undergoing resolution into their primitive elements, hydrocarbons and ammonia. Wagner even enclosed albuminous substances in the peritoneal cavity, and found them undergo this fatty degeneration. It would appear indeed that this is the retrograde tissue metamorphosis of sarcous elements, the true histolysis of muscular fibre when it is no longer renewed from the germinal matter; no matter how that is brought about, whether it is due to incomplete or imperfect blood supply, or the arrest is due to death.

It would appear that the non-renewal of tissue from the germinal matter is the cause (or, if not that exactly, furnishes the circumstances under which it goes on) of this fatty histolysis. We now know from the researches of Lionel Beale chiefly, that growth is confined to the germinal matter, and that formed material merely fulfils its function, and then undergoes molecular histolysis; the place of the worn-out material, or the material which has run its course, being filled by new formed material from this germinal matter. The knowledge of this would prepare us for the fact that fatty degeneration of the muscular structure of the heart commences in the nuclei, in the germinal matter. Paget observed granular degeneration in the nuclei at a very early stage, and a reference to the plate above will show that the fatty particles are found first around and in the neighbourhood of these nuclei. When examined under the microscope, the first changes will be found to consist of the appearance of dark clots, not unlike the pigment granules of brown atrophy of the heart; these are stated by von Dusch to consist of the colouring material of the muscular fibre. This is not inconsistent with what we know of the change of colour in the frog's skin, which is achieved by the diffusing and gathering together of the pigment matter. When diffused a dark colour is given, when aggregated in little masses a lighter colour is produced. So in the muscle of the heart, it is pale and colourless, and its colouring material is found in diminu-

tive masses. The cross-markings, or striæ, of the muscular fibrillæ appear faint, and further on become lost. The dark clots enlarge, and become transparent, and are seen as globules of fat, which Dr. Quain says "are extremely like the oil-globules of milk, and appear to have a mere albuminous envelope." Still later they have increased in number and size, till the muscular fibril with its cross-markings is lost, and the sarcolemma contains merely a number of fat globules in rows, or, as Rokitansky has described them, "like strings of beads." The process does not pass on by continuity from one muscular bundle to another, or even from one fibril to another in a bundle, but is irregular; and side by side may be seen fibres in all conditions, from the still normal to the most advanced condition, with fibrils in various stages among them.

During this process the muscular fibre has been undergoing naked-eye changes, and changes in consistency. The colour has become paler, for the reason given above, and is of a dirty yellow, or of a "dead-leaf" colour, somewhat unevenly though, and some parts are paler than others. The consistency is altered, the wall breaks down readily under the finger, and a finger may, in advanced cases, be pushed through the ventricular wall, as if through one or two thicknesses of wetted paper. The tissue is friable, and tears with a sort of fracture, and crumbles down under the finger and thumb. It feels greasy and unlike normal heart-wall, and a warm knife passed through it looks oily. There is not necessarily any change in bulk, nor does the degenerate structure take on dilatation inevitably. The change may be localised and confined to some particular portion, especially when due to disease of the coronary arteries. This may give a mottled appearance to the heart, but as commonly, and more so, the heart is generally of this pale-buff colour, the blue coronary veins looking marked and distinct. These veins with their deep blue form a striking contrast to the paler tissue around them, and are often themselves tortuous and atheromatous. Nor is there necessarily a deposit of fat along the sulcus in which the veins lie, which makes the contrast in colour striking. The coronary arteries are not uncommonly tortu-

ous, rigid, and atheromatous. The whole appearance in a well-marked case is that of degeneration, of partial death, or imperfectly renewed life, which is quite borne out by the microscopic appearances. It must not, however, be supposed that we are yet in possession of all that we can hope to know of this interesting form of histolysis, and much must be added to what we yet know ere our knowledge even approximates completeness; but such as is known is fairly certainly ascertained, and may be relied upon. It has been conjectured that this degeneration more readily occurs in those new fibres which arise in hyperplasia, commonly called hypertrophy; that the new fibres more readily take on degeneration than the original ones, but of this there is no proof; any analogy founded on the more ready decay of new growth than normal tissue, does not hold good in hyperplasia, where the new fibres are as perfect as the original ones.

Causation.—The causation of fatty degeneration of the muscular fibrillæ of the heart is yet so unascertained, that the circumstances under which it occurs must be supposited for it, with such inferences as they may appear to indicate. The presence of it where the blood supply of a part of the heart is cut off, partially or more completely, shows that it is closely allied to the deprivation of blood, and thus the non-removal of worn-out material, as well as the non-supply of new tissue. Thus it will occur in an intense degree in the part supplied by an arterial twig when that vessel is choked with an embolon, whether, in acute disease, as an embolon from gangrenous lungs, or consisting of a small mass of atheroma washed off some atheromatous tubercle, or out of an atheromatous patch, breaking up and forming an ulcer in the arterial wall. It is, as a more general and diffused, but very chronic and gradual, condition, associated with an atheromatous condition of the arterial system. Here it has been described as a preservative lesion. Sir Wm. Jenner, in his address before the British Medical Association, at Leeds, in 1869, stated that when this announcement was first made in the Pathological Society of London, it was received with shouts of laughter. But as knowledge on the subject became more general, it was found that the statement was strictly

founded on fact. There is no doubt that when the arteries become atheromatous and rotten, an unimpaired heart would be a most unquestionable source of danger, and great danger, while an impaired condition of the heart avoids the risks and is thus preservative; though, of course, it must be owned that it substitutes new risks in itself for the other risks it obviates. That, however, a fair balance of power between a degenerate heart and degenerate arteries gives a better probability of temporary continuation of life than if the arteries were exposed to the shock of a perfectly normal heart, will not be questioned by many clinical observers. This association betwixt fatty degeneration of the heart and atheromatous arteries is not, however, accidental, and the consideration of the association may not be either out of place, or uninstructive. The heart, as we saw in the opening chapter, is dependent for its blood supply on the arterial or aortic recoil. It thus receives its blood supply during its period of sleep, of muscular quiet, when the flow is not impeded, as would certainly be the case were it to receive its blood supply from the ventricular systole, its own period of activity. The amount of aortic recoil, then, affects the blood supply through the coronary arteries. When from any cause there is increased arterial tension, there is increased aortic recoil and freer supply of blood to the heart itself. Thus hypertrophy tends to maintain itself; dilatation tends to defective blood supply and heart degeneration. These causes affecting the heart nutrition through its blood supply, are quite distinct from any vaso-motor variation which may be carried on through the cardiac ganglia, and depending on impressions made on them, probably by the sensation of distension. When, then, we have an hypertrophied heart pretty forcibly dilating the aorta, and thus leading to good recoil, there is good blood supply to its own structure. But, let that strong recoil lead to aortic valvulitis and insufficiency, so that the blood is no longer arrested on its backward course perfectly, and thus the coronary vessels filled, and we find, as Mauriac has shown, that that hypertrophied muscular wall is rapidly affected by fatty degeneration. So, too, when the arteries, and especially the aorta, are, as a consequence

of this powerful distension, themselves becoming atheromatous, they then become dilated instead of recoiling perfectly on each systole, they become less elastic, and their recoil is imperfect; then the imperfect recoil leads to insufficient blood supply to the coronary arteries and impaired nutrition of the heart itself. Thus we see that as time rolls on the atheromatous arteries and the degenerate heart become adapted in power to each other, and a preservative balancing of forces tends directly to the continuation of the existence of the organism. The weakened heart tests less forcibly the diseased arterial coats, and thus the risk of rupture of them is lessened. But as that risk is lessened, unquestionably other risks are supposited for them, and the danger of death from a ruptured artery ultimately is lost in the gradual oncoming of inevitable cardiac cessation in diastole. Thus we often see families where certain members die at an early old age with apoplexy, while those who survive to an advanced old age die of cardiac failure. Apoplexy is the danger of the first stage of hypertrophied heart and hardening arteries; while death from heart failure is the almost inevitable end of the more advanced condition. Thus at first the more rigid arteries obstruct the flow of blood, and the heart becomes hypertrophied; but this compensatory hyperplasia is not without risks, as consequence of itself. The increased ventricular force may, and often does, rupture one of the rigid arteries, and more especially if the degeneration in the arterial coats be unequal, and one part is less equal to the increased tension than another. Or it may take place where the vessels are unusually thin-walled, as in the brain, and so most readily give to the increased tension; thus cerebral apoplexy is very commonly associated with hypertrophied heart, as Rokitansky has shown. But in time things alter, and the heart itself becomes the great source of danger, and what the patient has gained on the one hand he has lost on another. No doubt for a time that cardiac impairment is undoubtedly preservative, but at length it becomes itself the great source of danger. There is ever danger, in time, of a compensatory process, preservative though it be, becoming itself the great danger to life; thus

in uræmia diarrhœa is compensatory and preservative, but nevertheless it is not free from danger.

The theory of compensation in morbid conditions has been pushed by incautious advocates and rashly accepted by non-thinkers till there is some risk at present of its being altogether forgotten that morbid processes, and not physiological actions, are being considered. Though the process may be largely physiological, it must not be forgotten that is the physiology of disease, if the expression may be permitted. So fatty degeneration of the heart is, when combined with, and probably taking its origin in, atheromatous arteries, a distinctly preservative lesion, but it none the less becomes in time the great danger itself. Still it is, like all compensatory actions, the substitution of a lesser evil for a greater. For if the vessels, as they are found on death by cardiac failure, had been all along subjected to the force of a normal heart, a rupture must almost inevitably have occurred before the date of the cardiac cessation. For the diminution of the distending force has slowed the diseased action in those arteries, and had those diseased arteries been all along subjected to the same distending force, as induced the disease originally, their decay must have been accelerated, and rupture of them unavertible. Thus the gain by this preservative action is unquestionable, at the same time we must admit that it cannot be regarded as an unalloyed good. This degenerative process in the circulatory system is of the greatest interest to the profession, and a knowledge of the various stages in it of the highest importance in our dealings with persons of advanced life, when the maintenance of life is becoming of great importance not only to the person, but to society, from the ripened intellect. This change from hypertrophy to fatty degeneration so profoundly modifies and alters the symptoms, that the varying signs and symptoms will have to be considered in a later section at some length, and in close connection with their pathological import, and the morbid processes indicated by them. Hypertrophy when not connected with altered arterial conditions, as in aortic obstruction, will remain unaltered for years; and the new tissue does not have any inferior or defective vitality, in con-

sequence of which it soon undergoes degeneration; the degeneration is as physiological as the hypertrophy, and has a similar, indeed an identical origin. This somewhat lengthy discussion may, perhaps, be useful and excusable, as helping to elucidate a subject, however familiar to some readers, not yet fully understood by the bulk of the profession.

Fatty degeneration is at other times connected with ossification of the coronary arteries. And it must be admitted that in some cases the coronary circulation is found in an extensively diseased condition, without the systemic circulation being to any like extent affected. This is a subject well worthy of being taken up by some youthful reader and carefully investigating. It is certainly a difficult subject, and as yet shrouded in mystery. It would appear, however, that the calibre of the first portion of the coronary arteries, and the construction of the orifice, by which they are exposed to unusual strain, offer an *à priori* probable clue. The coronary circulation is not, however, itself always equally affected, and some parts are more diseased than others, leading to more and less advanced conditions of degeneration in the muscular walls themselves. The coronary vessels are often obstructed by growths of atheroma in tubercular masses at their orifice, and sudden blocking by a dislodged mass is one cause of sudden death by cardiac syncope.

Fatty degeneration, too, as connected with imperfect blood supply, may arise from partial obliteration of the coronary arteries by pericardial adhesion along or across them; for the sulci in which they run are not deep enough to completely protect them from this danger.

Where this degeneration is the result of disease, &c., of the coronary vessels, it may be limited to the right or left side according to the artery affected; or even be more localised when a branch is diseased or plugged by an embolon.

Fatty degeneration is also one of the risks which dog cardiac dilatation, for this imperfect arterial distension, produced from feeble ventricular contraction, leads to imperfect arterial recoil and impaired tissue nutrition. From these various considerations we see that fatty degeneration of the heart is

intimately associated with defective blood supply, and that its course is acute, or chronic, in direct proportion to the diminution in the blood supply: where cut off abruptly, the degeneration is rapid and pronounced; where a gradual diminution has slowly been instituted, the degeneration is more protracted.

There are, however, conditions which originate or induce this muscular degeneration, which are not intimately associated with the amount of blood supply. This fatty degeneration of the heart has been found allied with a degenerative condition of the lungs and kidneys, in which general degenerated condition the disease in each organ goes on hand in hand with each other; not, however, standing to each other in the relation of cause and effect, but apparently each the result of some general condition common to each as a provoking cause. This compound state of visceral degeneration has been described by Beale and Basham, and the latter in his work on "Dropsy" gives some most interesting cases illustrative of it, and also by R. Virchow, who also found an impaired and degenerate condition of the gastric mucous lining to accompany it. It has been supposed that this degeneration may be due to some defect in the trophic nerves in these viscera, taking its rise in some impaired condition of the organic system of nerves, some derangement in what Bichat denominated the vegetative processes, but, as yet, this is merely speculative.

The connection of fatty degeneration of the heart with sclerosis of the cardiac ganglia, or other disease of them, is merely presumed: the subject is no more than an inviting field for original investigation.

There is still one more curious matter about this muscular degeneration in relation to its causation, and which throws a side light upon its pathology, and that is its relation to poisoning. Not only did E. Wagner find it in connection with phosphorus poisoning, but it has also been found in poisoning with other acids than phosphoric. Von Dusch gives a list of sulphuric, nitric, phosphoric, oxalic, and tartaric acids, coupled with the names of Koch, Rokitansky, Lewin, Tüngel, E. Wagner, Löwe, Mannkopf, Munk, and

Leyden. This curious association of it with poisoning by acids is apparently somehow connected with the evolution of nitrogen in the process of this fatty degeneration of albuminous compounds, as first observed by Michaelis. Whether these acids when administered in excess, circulate in the blood, and by depriving the nitrogenised tissues of their ammonia, and thus leaving the other constituents as fat, or not, we cannot tell. If this should be found to be the case, it will increase the importance of the alkalinity of the blood, as protecting the muscular tissues. The tissues are always impaired in those deeply sœptic pyœmic conditions in which Virchow found the blood positively acid. That in this condition, as well as the poisoning by acids, the heart's structure should be so deeply involved, is explained by its free blood supply, as compared to other muscular structures, and not by any peculiarity in itself.

From these considerations we can surmise that this fatty degeneration is a histolytic process, variously instituted. That it commences in the germinal matter, and spreads therefrom into the formed material, gives us some idea of its being an arrest in nutritive processes, seen first where the processes are conducted, and gradually found more and more extensively as the formed material wears out, in the performance of its function, and is no longer perfectly renewed. Further than this we cannot go, nor will any consideration of the pathology, so far as yet known, aid us in any way in affecting this morbid process therapeutically, except so far as it is connected with the amount and nature of the blood supply. Its relation, however, to anœmic or spanœmic conditions is not yet proved to be such as to warrant any inference that it ordinarily, if ever, takes its origin in them.

Age.—The age at which this affection is most liable to show itself is certainly that of advanced life; the greatest number of deaths from fatty degeneration occurring in any one year is stated by Sir Thomas Watson to be in the 63rd year. There is no doubt that it is a comparatively rare affection in early life; when it occurs then, it is almost always in consequence of its accompanying some general affection, or from pericardial adhesion. Its frequency in-

creases in middle life, and attains its maximum at or about the age fixed by Watson, its comparative frequency diminishing as the more advanced periods of old age are reached. But in considering this question, it is not possible to speak with any exactness, and an array of statistics would add little to the importance to be attached to any more positive statement. Many cases are obscured by other lesions, and the majority of causes of death are not corroborated by *post mortem* examination.

Sex.— There is every reason to suppose that the male sex are more liable to fatty degeneration of the heart than the female sex, but not by any remarkable preponderance. (Dr. Quain, however, calculates it as 5 to 2, males being much more liable to it.) It would not be correct to suppose that every member of the weaker sex who makes ostentatious display of "a weak heart," is positively suffering from any structural affection of the heart walls, even when occasional attacks of anjina are present. But there is grave ground for suspicion in those numerous cases, especially in those whose lot does not subject them to exertion, for that would soon test the truth, when at or near the menopause a lady is noticed to be short of breath, blue about the lips, the countenance at times injected, vertigo, and syncope not unusual, and induced by slight exertion, the feet cold, and the hands white, puffy-looking, and pasty. The sounds of the heart are thin but clear, and the pulse feeble and compressible, and the amount of urine scanty, and laden with lithates. If, in addition, the first sound is out of all proportion slight as compared to the second sound, which, though not accentuated, is clear and distinct; if the first sound is almost lost, or merely consists of the sound occasioned by the closure of the auriculo-ventricular valves, there is grave ground, indeed, for suspicion. But the numerous cases where, after the critical period has been passed, the symptoms vanish, demonstrate that though these symptoms are serious, they may indicate some other affection of the heart walls than this especial form of degeneration of the muscular fibrillæ. This is somewhat a digression from the exact subject under consideration, and yet it bears on, not exactly the comparative frequency

in the two sexes, but what we think in our experience is the comparative frequency. It is very obvious that in the absence of more general *post mortem* examination, this, as well as some other matters of fact, will remain to a great extent a matter of opinion.

Occupation, &c.—It is said that fatty degeneration is a common affection among London gin-drinkers, and it has certainly appeared to me a common complaint among elderly publicans. How far, when so occurring, it is a consequence of the constant existence on a hydro-carbonaceous food, for many of these persons can scarcely be said to eat, which is unsuited to repair of a constantly wearing out nitrogenised tissue, or not, it is not possible to say with any certainty. It is, perhaps, too, somewhat common among men whose pursuits entail very little or no bodily exercise, and it has been stated to be a common cause of death to judges. How far the position and power to obtain large amounts of rich food without much bodily exertion, leads to an atheromatous condition of the vessels, which in its turn entails a fatty degeneration of the heart, is the cause of this disease; or how far the respired oxygen is met and neutralised by the hydrocarbons in the blood, and thus is not available for the removal of worn out waste material, a necessary preliminary to the growth of new tissue, are subjects yet uninvestigated. The non-development of muscular tissue on a purely hydrocarbonaceous diet, and the imperfect removal of waste material in those who live well and work little, must not be confounded with the development of fat cells in the intermuscular tissue (Neufettbildung), often associated with general development of adipose tissue. General fatness is no protection against fatty degeneration of the heart, but neither is extreme leanness. These persons die as much from this degenerative tissue-decay as do stout persons; and it may be found in persons of every position in life, from the millionaire to the bricklayer's labourer.

Physical Signs.—The physical signs are more decidedly negative than positive, for this degenerative decay is marked not by positive manifestations, but by failure of ordinary manifestations; the absence of action and power.

Inspection.—There is nothing to inspection but the absence of action, and even in those thin persons where normally a movement over the apex-beat can be seen, especially with the light falling on it, no movement is detectable.

Palpation.—On placing the hand over the heart no movement is felt, if the degeneration has commenced in a heart of normal size. But if, as not unfrequently happens, there is degeneration setting in in a heart which has previously been hypertrophied, there is the tumbling, rolling, irregular action, very similar to a weak dilated heart, with a sensation of diffusion, as if the apex were broadened; indeed we know that the process of dilatation goes on when hypertrophy is yielding to this tissue-necrosis. Still, these positive symptoms are not associated with the fatty degeneration, but with the hypertrophy. It may indeed be gravely questioned if ever, even under the most probable circumstances, any excited action be felt in a degenerate heart. There is no more laborious action in the heart than there is its outward visible sign palpitation; instead of that there is almost total abolition of action and syncope.

Percussion.—Neither is there increase of percussion dulness. Dilatation of the degenerate heart is not a characteristic of this form of disease. Where dilatation is most usually found, is when hypertrophy has already existed and is yielding. The degenerate heart not previously altered in bulk or shape, does not usually become dilated: it may, however, do so. There is not then usually, or necessarily, any increase in percussion dulness. Nor can any alteration in the bulk of the heart be regarded as characteristic of fatty degeneration; neither diminution nor enlargement belong to it in itself. It is highly questionable whether this structural decay leads to any lessening of the heart's bulk, any atrophy of it (though the diseases have been regarded as associated), and though it is found in hearts both hypertrophied and dilated; still neither of these changes can be considered to be allied with it. Though dilatation will result from it in a heart previously hypertrophied, and any change in shape in an hypertrophied heart is significant, along with other indications, still we are not entitled to regard dilatation as an

essential characteristic of fatty degeneration; though it may result from it both in normal as well as hypertrophied hearts.

Auscultation. To the negative results furnished by palpation chiefly, we must add the negative and positive ones yielded to auscultation. This last is our most trustworthy guide in forming a diagnosis of fatty degeneration. The first and most absolutely positive indication is diminution of the first sound. The first sound may, in advanced cases, be almost absolutely lost, and, in comparison with the second sound, is inaudible. In less marked cases the first sound may be very faint, and is obscure compared to the clear thin second sound. When the first sound is, as it not uncommonly is, almost entirely due to the closure of the auriculo-ventricular valves, it is thin and clear, and much resembles the second sound. In every case, indeed, the muscular sound is impaired; the muscular portion of the first sound is absent, more or less completely. Thus the first sound wants its ordinary duration, its chronicity as compared to the second sound; the heart's sounds are almost those of two second sounds following each other. This loss of the first sound is the most valuable single sign in connection with the disease under consideration.

But when there has been previously existing hypertrophy there may still exist a fair amount of muscular sound from the amount of muscular fibre remaining healthy. Then it comes to a question of comparison of sound with bulk and shape, a question which must necessarily rest on each man's individual experience, not, indeed, about it itself, but about his power to appraise it. There is no more difficult diagnosis, not even in abdominal tumours, than in this combination of pre-existing hypertrophy and more recent degeneration. The subjective and objective symptoms, the patient's history, his failing power may throw some light on the complication in the physical signs, but the progress of the case and repeated examination alone will decide it, and even then allowances must be made for the possibility of temporary improvement under remedies and suitable treatment.

The characteristic sign of fatty degeneration is impair-

ment or loss of first sound; a condition, however, which will occur in elderly persons in anœmic states, and passes away with the condition on which it causatively depends. Thus loss of first sound must not merely be detected, but the observations must be repeated, and it must be found to be persistent, then it is of the gravest import: but temporary loss or impairment of first sound may be found along with other conditions than that of structural degeneration of the heart-walls.

Impairment of first sound is the characteristic of disease of the muscular walls of the heart, and though most commonly found allied to fatty degeneration of them, for the obvious reason that this is the most common affection, is also found along with fatty interstitial infiltration, amyloid heart, &c. Indeed the signs of fatty heart are shared by those other conditions, which preserve both the signs, and, to some extent, the symptoms of this affection.

Objective Symptoms.—These are not confined to the heart, but extend to the arterial system; and the changes formed in the eye and skin may also correctly be classed under this head.

Objective Symptoms in the Heart itself.—Firstly, we put aside palpitation. Palpitation is not a symptom of disease of the heart walls. The heart walls when diseased, and especially when degenerate, do not act laboriously ordinarily, and consequently there is not palpitation. Palpitation is rather the characteristic of distension and dilatation in a heart structurally sound. The degenerate heart does not give out this active witness of inability; instead of that there is syncope, more or less perfect. Positive acknowledgment of inability is the characteristic of cardiac degeneration. The sound heart may be distended and try to labour so handicapped, but the structurally diseased heart will not distend, but suffers almost complete cessation of action when exposed to such action as would induce distension and palpitation in a structurally sound but dilated heart. True, when there is previous hypertrophy there is distension, and the resumption of that dilating process, which hypertrophy arrested, and, consequently, palpitation. Still, we are

entitled to regard palpitation as associated with an over-distended or dilated heart, and not as associated with fatty degeneration. It may occur in the complex condition of hypertrophy undergoing degeneration and becoming dilated, but it is with the dilatation of the sound fibres, not the decay of the diseased ones that it is associated. Palpitation is the characteristic of dilatation, syncope of structural degeneration.

Irregularity.—Irregularity or arrested, delayed, ventricular systole is associated with structural degeneration as well as other affections of the heart, valvular or dilated conditions. It is the irregularity occurring at intervals, a number of normal beats, both in time and volume, and then a pause, often preceded by two or three rapid beats. Or again, with an undulating, feeble pulse, with prolonged halts, that is very often met with in fatty degeneration. The irregularity is increased by any effort, going upstairs, difficulty in emptying the bowels, or the physical excitement often arising from mental disturbance. The increase in amount of irregularity on effort, or any call on the heart, is ever indicative of organic disease. The effect of effort will always almost settle the diagnosis betwixt the irregularity of organic disease and those purely nervous disturbances so similar, indeed identical, for a full and able explanation of which the profession owes its thanks to B. W. Richardson. The effect of effort will almost alone decide the question, without appeal to the subjective symptoms, rarely or never absent in the one case, and in no way associated with the other. In a dilated heart with persistent irregularity, effort usually induces palpitation as a temporary condition implanted on the permanent one; be that dilatation connected with other conditions as it may; but in structural degeneration there are more absolute evidences of failure of power. Instead of palpitation, the irregularity is increased, and becomes more marked, and the ominous halt of intermittency is rather found than any such positive action as is implied by palpitation.

Intermittency.—In fatty degeneration of the muscular fibrillæ of the heart, there are commonly found halts, more

prolonged than is compatible with the term irregularity: the pause is so pronounced as to establish its claim to the term intermittency, *i.e.*, there is intermittent action, a pause over the period of one ventricular contraction. This may extend over one or more auricular contractions,* and be more or less pronounced. It may occur at almost rhythmical intervals, the periods being shortened by effort or excitement. It has been disputed whether during this pause the ventricle halted, or whether a contraction took place too imperfect to produce any perceptible effect on the radial pulse.

Personally, I am inclined to think that both conditions occur, and in some cases on applying a stethoscope over the heart, while the finger is kept on the radial pulse, during the pauses a feeble ventricular contraction may be heard but not felt. Nor is this surprising! A feeble ventricular contraction, and so perceptibly imperfect to the ear, would lose most of its effect on the backward flow occurring before the auriculo-ventricular valve is closed, and little even of its feeble impulse be transmitted to the radial pulse. This view is corroborated by the character of the second sound, which is quite, or all but, inaudible. There is reason for supposing that such imperfect ventricular systole may occur as will leave no impression on the radial artery. But the question is not a practically important one. The prolonged ventricular halt amidst the other evidences of failing power in the circulation is ever prognostically grave, and when slight effort induces this prolonged halt amidst the flutter of irregularity, its presence is ever ominous. Sometimes this halt is found along with an ordinarily slow pulse, a pulse not so quick as the normal: indeed a rapid pulse is not characteristic of fatty degeneration, but rather a moderately quiet, slow, feeble pulse.

Not very rarely the halt will come consecutively for two beats or more, and then the rythm of the heart is much disturbed. Disturbance of rythm, by delay, combined with loss of power, is almost characteristic of fatty degeneration,

* See a paper by the writer in the *Lancet* of April 13th, 1872, on "Cardiac Intermittency," for the evidence of this ventricular pause over one or more auricular contractions.

especially when associated with loss of first sound. When so accompanied, disturbance of rythm is not nervous but muscular, and the condition indicated not nervous disturbance, but structural disease of the walls of the heart.

In the Arteries.—There is not necessarily atheroma of the arteries, though it is usually present. The pulse is feeble, compressible, unsustained, and easily obliterated by pressure. It is the reflex of the heart, and the elastic arterial system merely reflects the impression made on it. Thus the ventricular contractions are repeated as received, a slight tardiness in the arterial pulse being observable. When irregularity is present in the heart's action, it is found in the pulse at the wrist, and as fatty degeneration is associated with the heart generally, and when localised not usually found in the right ventricle, there is usually a corresponding irregularity, and not a more marked one to auscultation than to the finger on the radial pulse. As, however, clinically, fatty heart and atheromatous arteries go commonly together, there is usually seen a tortuous temporal artery meandering away along a temple wrinkled and bald. Sometimes the artery is unusually prominent and covered with a parchment-like tegument, while the temples are either covered with grey hair, where the hair is coarse and strong, or a few fine grey, silvery hairs may be found far back, with a perfectly bald crown. When the arteries are atheromatous, a totally false impression of strength may be communicated by the pulse to the finger, and this falseness is detected in auscultating the heart.

The Eye.—Much discussion has taken place since E. Canton first gave his opinion on the diagnostic value of the "Arcus Senilis," as indicative of fatty degeneration of the heart. Its importance, however, as an auxiliary to diagnosis, is now generally admitted. Its presence alone is not sufficient to warrant even the most elementary diagnosis. In fact, this opaque ring at the union of the sclerotic and the cornea, is not always truly fatty, in a degenerative sense. It may be found as a clear, bluish ring, giving a peculiar expression to an eye still bright and vivacious.

The owner of this eye may be old or elderly, but is active in mind and body, and it is often found allied to unusually

hale, active old age; green old age, in fact, both mentally as well as corporeally. This ring is not of bad prognostic import. It is perhaps as much calcareous as fatty, and is homologous with the osseous ring found normally here in the bird.

But at other times this ring is found yellowish, with ill-defined edges, with a dim eye, a somewhat tottering or tremulous gait, and a muscular unsteadiness, and when so associated, its prognostic as well as its diagnostic import is widely different. When this ring is indistinct about its edges, and gradualiy melts away into a cloudy cornea, it is pathognomonic of degenerative changes. The cloudiness of cornea is due to the presence of globules of fat scattered throughout its substance, to real degeneration of structure, and this cloudiness gives the dim look to the eye, and often a doubtful, wavering expression, which is heightened by the hesitating manner and muscular unsteadiness. This eye is never, perhaps absolutely never, found without a shaky pulse, a feeble heart, and great evidence of general decadence. The importance of this objective symptom must not be underrated, as it frequently attracts attention to the general condition, which might otherwise not be evoked by the patient's appearance or complaints. But the two forms of arcus senilis must not be forgotten.

The Skin.—A field is open here to some exact clinical observations, combined with microscopical observation. The skin in many cases of cardiac degeneration is distinctly altered to the naked eye. The appearance sometimes is that of discoloured parchment, while at other times the epidermis looks thick, opaque, and dull, there is none of the glistening which is not altogether wanting in the healthy skin, even of the aged. It is not the dry, harsh skin of mania, or even of chronic Bright's disease, where the obstructed circulation, through the thickened arterioles, leads to a dry, hard, unelastic skin. At other times the skin is greasy to the feel like leather, or the outside of a bladder of lard, and the epidermal cells rub off, looking fatty and degenerate.

Along with Dr. Clifford Allbutt, the writer often observed these changes in the skin in patients, furnishing evidence of

degenerative changes in the viscera, and especially in those suffering from fatty degeneration of the heart. There is, however, opportunity for more exact observations than those we made, which were confined to the naked eye appearances and to the sensations elicited by touch. The degenerative processes extended to the hair which, if fine and silken, usually dies of necrosis, or only a thin fringe extends from the temples round the occiput. When it is naturally strong and coarse it remains, and though "a hoary head is a crown of glory," if the working of it be what it should be, still we find that there is a degenerative process going on in it, allied to that going on in degenerate heart fibres, that is, that the loss of colour is due to the collecting of the pigment in small masses, which can be seen under the microscope along the axis of the hair.

Subjective Symptoms.—The subjective symptoms of fatty degeneration are pronounced and positive, and present in the great majority of cases. They consist of breathlessness, syncope, angina pectoris, cerebral anœmia, muscular tremulousness, impaired mental action, &c.

Breathlessness is most commonly and certainly present. It differs from the difficult respiration of cardiac asthma, or pulmonary congestion with dilated right ventricle. It is shallow, hurried, without effort, while the face is pale, free from congestion, and has a look of wild alarm very often; in cardiac dyspnœa there is heavy, imperfect, laborious breathing, often with bronchial whistling, or moist rales, and the countenance is injected, the lips blue, and the stamp on the features is that of physical distress.

Syncope.—This is commonly found during the course of fatty degeneration, and is induced by effort. When a sudden effort or exertion is made, an obstruction is offered to the flow of blood, the call upon the impaired heart is increased, and its inability reveals itself in syncope. This sudden heart failure is characteristic, and differs from the sudden death merely in degree. Death is prolonged syncope, or may be rupture.

This syncope, and even death, is not uncommonly met with in elderly persons who long have felt themselves less

equal to exertion, but, with that disinclination to think themselves failing in power, let alone making mention of it to any one, which characterises the oncome of age, have thought as little as possible of it, when some sudden exertion is ventured upon. Thus hurry to catch a train, or even more, haste to catch an omnibus, with the anxiety, as a stimulus to exertion, increased by its visible propinquity, and not rarely by the conductor who will allow it to move on a short way in order to give the lagging would-be passenger a lesson, not uncommonly induce an effort which results in most distressing faintness and general uneasiness, sometimes lasting for hours or even in sudden death.

There is some reason for thinking that some effort usually precedes those sudden deaths, without apparent exciting cause, but of course there is no means of interrogating the dead, or eliciting the facts. But sudden failure does not arise spontaneously usually: if all the circumstances could be ascertained, some exciting cause, may be a comparatively trifling one, would be elicited. Effort on the night-chair notoriously produces alarming faintness, and not rarely sudden death. The sudden assumption of the erect posture, by throwing the weight of the column of blood in the ascending aorta and carotids, &c., in the enfeebled heart, will also produce both temporary and persistent syncope.

Angina Pectoris.—This symptom was long considered pathognomonic of fatty degeneration. It is not so however, though it is, as a symptom, often so associated. Angina pectoris may, like the true objective symptoms in the heart, be either nervous derangement, or associated with structural histo-necrosis. The two forms of angina are closely similar if not identical, in their symptomatology, and the general collateral symptoms and signs must be called in to establish the diagnosis. As, however, angina pectoris will be considered separately, as a nervous affection, it is not necessary here to do more than give it as found when accompanying fatty degeneration.

The attack is usually sudden, the chest seems held in a vice, the breathing becomes shallow, not laboured, muscular power seems paralysed, the features become stamped with a

combination of alarm and horror, the face is pale, and down the brow roll large bead-like drops of perspiration, and the voice becomes weak and importunate. There is, or is not, pain in the chest, though usually there is that sickening pain which is received through the sympathetic, when an impression on it is powerful enough to produce pain. The patient's condition excites the greatest alarm and sympathy in onlookers, who, no less than the patient, are apprehensive of instant dissolution occurring. This condition may recur several times, or the first may be fatal. After it the patient is left prostrate, shaken, and unnerved for some time. If the attack be nervous, in a day or two at any rate, the heart sounds will be found normal and good, when it is connected with fatty degeneration there is feeble impulse, loss of first sound, &c., &c. W. T. Gairdiner has described a form of angina sine dolore, in which all the symptoms are present, except pain, which is not essential to it. This angina pectoris must be discriminated from the attacks of cardiac dyspnœa, which are somewhat similar, and have been termed false angina pectoris. Cardiac dyspnœa is usually accompanied by injected countenance, the absence of the bead-like drops of sweat, though they may be present, blue lips, all the evidences indeed of passing cyanosis, while the respiration is laborious, obviously and evidently connected with effort, and there are chest rales and other evidences to the stethoscope of pulmonary congestion, as loud, harsh respiration, &c., and the assumption by the patient of the sitting posture even when in bed. The diagnosis of it, and that form of cardiac apnœa which is associated with spasm of the pulmonic arterioles and thus arrest of respiration is more complicated, but in this latter there is also cyanosis and right side distension, &c. The diagnosis betwixt angina and asthma need not be described. In all these cases the pulse is feeble, irregular, and almost imperceptible. But in angina the heart sounds are equally feeble and the apex beat undetectable, while in those cases where the obstruction in the lungs leads to right side distension, there is palpitation, as the evidence of laboriousness, in the right heart: indeed, Hope says that the most violent palpitation is often found when the right

ventricle is distended almost to paralysis in aggravated cardiac dyspnœa. In angina pectoris, there is no action of the auxiliary muscles of respiration, which are always in great, and even violent, action in the other forms of disease with which it may be confounded. In the other forms there is obvious difficulty in breathing: in angina pectoris there is rather an abolition of the act. The pathology of angina will be given in the section on it in Chapter XII.

There is not in fatty degeneration the oppression in the chest, the sensation of labouring, which occurs in other cases where disturbance of respiration is associated with other heart affections. There is rather an avoidance of effort, an instinctive spontaneous shirking from effort in fatty degeneration, than the voluntary intelligent avoidance of effort, in consequence of the difficult respiration it induces, as experience has taught the patient, which is the cause of quiet in the other forms of heart disease.

Cerebral Anœmia.—This is a condition which is frequently associated with disease of the heart, and therefore with fatty degeneration. The imperfect circulation deprives the brain of its supply of arterial blood, and the chronic imperfect supply may cause a condition of chronic cerebral anœmia. It may, however, be subject to occasional acute exacerbations, much resembling an apoplectic form of attack. There is loss of consciousness and muscular power, somewhat more sudden than the more gradual oncome of apoplexy; there is a small, slow pulse, with unfilled arteries, and cold, pale skin. This condition may persist for some hours even, and longer. In speaking of anœmic conditions of the brain, Todd, in his Cyclopædia of Anatomy and Physiology, article "Abnormal Anatomy of Nerve-Centres," says, "It (ænœmia) is also present when the heart, oppressed by some disease affecting its own structure, fails to propel the blood with its proper force into the brain." This effect upon the brain, of inability in the heart, produces not merely physical, but psychical manifestations even. B. W. Richardson, in his "Discourses on Practical Physic," (1871), article on "Intermittent Pulse and Palpitation," says, "The man or woman with a hesitating heart is thereby unfitted for sudden tasks, demands, resolves,

which, when the heart is firm, are considered as of comparatively little moment, for when the heart hesitates, the brain, which reposes for its power on the blood the heart supplies to it, falters with the heart, just as the gas flickers when the steady pressure is taken off the main. From these circumstances some persons, who once were known as resolute and determined, lose those qualities when they are subjected to intermittent action of the heart, becoming, as their friends say, uncertain and doubtful in character, becoming, as they themselves feel and know, less the masters of themselves, and less secure in their own work, and skill, and power." There is no doubt about the truthfulness of this picture, drawn, as it is, by a master hand.

The cerebral anœmia which accompanies fatty degeneration of the heart, has attracted attention in consequence of a peculiar effect upon respiration induced by it, called "the phenomenon of Cheyne." This accomplished Dublin physician first observed a peculiar ebb and flow in the respiration, a dying away of the respiration, till it became apparently lost, and then a gradual deepening of it again, till one or two deep respirations passed, and then a gradual fading away of it. This symptom has been noticed elsewhere, and need only be mentioned here in order that its diagnostic value may be given. It is not peculiar to fatty degeneration, and von Dusch has noticed it in tumours of the brain, in basilar-meningitis, in uræmic coma, and in a grave attack of pericarditis. It first struck the writer forcibly in a case of genuine apoplexy, and next in the venous congestion and sopor of primary tricuscipid regurgitation.

Muscular Tremulousness.—This imperfect supply of blood to the brain affects the muscular system generally, and gives an uncertainty to the step, and a tottering appearance to the gait, while sudden exacerbations occur, when the beat falters more than usual, in which the patient is seized by a sudden feeling of loss of power, and snatches convulsively at the nearest object, tree, or lamp-post, companion, or stair-rail. This sudden convulsive snatching is momentary, and passes off as quickly as it came, but the general enfeebled muscular power is persistent. There is a peculiar expression in the

gait of the person with cerebral anœmia from fatty heart, a sort of guarded, quiet, distrustful walk, an avoidance of shocks or effort, combined with evident unsteadiness; there is a want of firmness in the step and tread, corresponding to what Dr. Richardson has said of the mind, which distinguishes it from the other gaits produced by cerebral or spinal degeneration. There is no irregularity, no loss of co-ordinating power, no jerking of limbs, neither is there trailing of them, or inability to lift them, nor yet the faltering step and disarranged toilet, so characteristic of certain nervous diseases. There is no paralysis of the sphincters, nor trouble with the bladder. Indeed from lessened arterial tension the urine is scanty, and usually laden with lithates.

History.—The history of fatty degeneration is in the very great majority of instances insidious and unmarked, at other times some peculiar occurrence or accident may have elicited the fact of its presence, and not uncommonly that occurrence is a visit to an insurance examiner. The discovery of fatty degeneration is often some personally unpleasing incident, often, however, a private discovery of the patient's, and kept carefully to himself with great secretive reticence. The appearance and condition of a person with fatty degeneration is somewhat difficult to describe, and instead of attempting to give exact clinical cases, a description of some various typical forms may neither be uninteresting or uninstructive. A case of moderately decided heart degeneration presents somewhat the following signs and symptoms. The patient is at or over middle age, with a comfortable enjoyment of existence, when at rest, with quiet pulse and respiration, and an appearance of fair health. But exertion is instinctively avoided, and his locomotion is evidently connected with exertion, all excitement is avoided, or, in excitable temperaments, found to disagree. The pulse becomes irregular on exertion, and the respiration is hurried, but not laboured. The heart-sounds on auscultation are feeble, and there is loss of proportion betwixt the first and second sound.

A more advanced case will present rather this appearance: an anxious or uneasy-looking face, with dim eye, discoloured or dull skin, and a tortuous temporal artery, with a bald or

grey head, great breathlessness, and inability for exertion; a pulse small, irregular, and with frequent halts, or sometimes a pulse rather as a fluttering undulation, difficult locomotion, and impaired or enfeebled intellect. The heart sounds are those of two second sounds, or the first may be entirely inaudible. There is cough, and somewhat of expectoration, but it is not bronchitis, nor yet the free expectoration when the bronchial veins partake of the general venous congestion, but rather a sort of degeneration of the bronchial mucous membrane. There is often a querulous and petulant voice, and a childish impatience and irritability. The patient will be noticed when walking to pant and look distressed, frequently to rest, either sitting down, or leaning against a lamp-post or other support. By avoidance of effort, this condition is compatible with a longer continuation of life than might at first sight seem possible.

Another case will appear somewhat thus. A pallid countenance, a look of wornout-ness, breathlessness, small, indeed tiny pulse, or exaggerated by an atheromatous condition of the arteries, loss of appetite, loss of all sexual desire, general debility, and indeed, premature old age. There are urine tube casts, with fatty globules in them, loss of heart sounds and impulse, rapidly increasing emphysema from degenerate lungs; an appearance, indeed, of ripeness, of ripeness merging into rottenness, a widespread, molecular necrosis, the precursor of general somatic death. This condition has been described by different writers, and is, if not the condition, closely allied to the condition, denominated and described by Sir Henry Halford, as "climacteric decay." The subjects of it are somewhat advanced in years, but their decay is in advance of their years.

This affection, fatty histo-necrosis, occurs in connection with hypertrophy which is giving way and presents somewhat the following condition and symptoms:—

A person advanced in years feels less equal to exertion than before; he has attacks of difficulty of breathing, with palpitation easily induced, is more liable to feel indigestion, &c. His pulse is moderately full, but somewhat irregular, and it is compressible out of proportion to its

strength. The heart is found hypertrophied and dilated, with a tumultuous action felt over a considerable portion of the chest wall, but there is a disproportion betwixt the heart's impulse and the radial pulse, which is suggestive.

It is obvious that here a difficulty in diagnosis may arise. Is the condition of the heart due to hypertrophy undergoing degeneration? Or is it due to dilatation not yet surrounded by hypertrophy? As far perhaps as the condition of the heart is perceptible to us, it is equally possible that the tumultuous action, with comparatively feeble compressible pulse, may be due to either. The history, however, does not point to a past history of inability for exertion, palpitation, dyspnœa, &c., being recovered from, or being relieved of late, but a consciousness of ever-increasing inability, growing from day to day, a condition each day or week worse than before; this will aid to settle the diagnosis. If not established, a little time will clear it up. If it is dilatation, not yet hypertrophied, but undergoing the process, improvement will be found, and decided improvement, especially if a suitable treatment be adopted. But if it is hypertrophy undergoing this histo-necrosis, a slight alleviation may result from treatment for a little time, but soon the downward process is again in obvious action. The condition in a comparatively brief time will be found somewhat so. The patient is breathless, liable to attacks of dyspnœa, which now call out the muscles of forced respiration, and are accompanied by lividity of countenance; he is less equal to exertion than ever. His pulse is more irregular and inclined to halt, much aggravated by any slight effort; his sleep is disturbed, and his legs are puffy. His heart's sounds are less distinct, the first being weak and short, the second clear and distinct, and the normal proportion betwixt them is quite lost; the heart's action is more tumultuous, rolling, and unrythmical. His whole condition is growing hopeless, and death may be sudden, or life may continue till the symptoms common to the later stages of heart disease are reached; and venous congestion has enfeebled every organ, from his kidneys to his bronchial mucous membranes, and the sufferings from this venous congestion are added to the original troubles.

Progress and Terminations.—The tendency of this fatty degeneration of the muscular fibrillæ is certainly to advance. Depending as it mostly does on a gradually diminishing blood supply, or on some defect in the fine processes of vegetative life, whose presence can only be suspected, "detected like a subtle odour" rather than demonstrated, its tendency is to advance in the downward direction. This molecular-necrosis of the structure of the heart is an insidious decay, for which neither nature or art possess a remedy. The progress is continued until the circulation is diminished and obstructed, as much as it can be by any valvular disease, unless the patient be cut off by sudden death. This is very liable to occur from fatal syncope. Dr. Quain states that, "out of 68 cases, death occurred in 21 by syncope lethalis" (*Lancet,* "Lumleian Lectures," April 6, 1872). This is a proportion of nearly one-third. That this proportion should be so high can furnish no surprise, when we remember the lessons of the dead-house and the advanced state of disease in which hearts are found, where the patient had died of some other affection. This syncope is usually associated with some effort, not immediately at the time, but some short time, hours or so, before. Some families are more liable to die of syncope from fatty degeneration than others, and most unquestionably men are more liable to it than women. It is in women that the most prolonged and painful cases can and do take place. Many cases in men are distressing and prolonged enough, but in women there is often a pertinacious, even cat-like, tenacity to life, till every painful sequel of heart disease is reached, and death takes place from carbonic acid poisoning. Sudden death is a merciful provision in heart disease, however appalling it may appear to those in health and strength, and is often the sincere hope of the patient, who indeed feels the troubles more than the human frame can bear. But it is not so common certainly after the stage of positive invalidism is reached. While the patient is still able to move about, sudden death may be induced by effort, or may occur from a heavy supper, but after the disease has progressed until all exertion is physically impossible, sudden death is not so

liable; and, when it does occur, will be found associated with some species of effort, as emptying the bowels, changing the linen, or even moving the position in bed. A gradual general quiescence has ceased to tax the heart by sudden calls on it, and a general lowering approximating the condition of the heart permits of life being still maintained; consequently the condition gradually grows worse; the œdema, once confined to the ankles, creeps up to the knees into the thighs and external organs of generation; there is œdema of the lungs and effusion into the pleural sacs and pericardium; there is ascites; the amount of urine is greatly diminished, and the urine is intensely acid, laden with lithates, and growing albuminous; there is venous congestion of the contents of the encephalon, and coma; or, from this source, is added to the symptoms irritability, whimsicalness, and puerility, occasioned by inefficient supply of arterial blood; there are dreams at night, hideous and even worse than the waking troubles. Then comes on carbonic acid poisoning from defective oxidation, through the clogging of the bronchiæ by effusion from the bronchial veins,* the patient nods off in a doze, which is not true sleep, wakens with a start from a horrible dream; dreading these dreams, he tries to keep awake, but soon again drops off, and then the case wears on till death comes to the relief.

It is possible that where this tissue-decay has been associated with a diet too exclusively hydro-carbonaceous, and containing too little nitrogen for tissue nutrition, that a different diet with proper remedial measures may lead to an improved histo-genesis and improvement; but it is to be feared these cases are exceedingly rare, unfortunately too rare to affect the prognosis.

Prognosis.—From the pathology little can be expected but the gloomiest prognosis as to the ultimate result. The prognosis as to time may vary, and may contain some small ground for hope. But life is terribly insecure. Fatal syncope

* My friend, R. Shirra Gibb, first pointed out to me in Vienna the error of Rindfleisch and others in supposing the increased bronchial mucus to come from the congestion of the lungs, this is anatomically impossible; it comes from the bronchial veins which partake in the general venous congestion.

or rupture may be easily induced, may occur at any time, as long as effort may induce them. The inevitable end may be yet in the distance, but it is distinct enough. In forming a prognosis the diagnosis must first be carefully made and verified by several observations in the earlier cases; and only after repeated and careful examination should the prognosis be delivered. In the more advanced stages of disease the prognosis is less difficult; but if the patient be only then seen for the first time, caution is necessary. The astonishing manner in which sufferers from heart disease, with an advanced condition of dropsical and other sequelæ, will sometimes rally for a time, and thus sadly disturb a too confident prognosis, without, of course, ultimately disproving it, is somewhat startling.

Treatment.—There are points in connection with fatty degeneration of the walls of the heart which justify some special allusion to them, in addition to the chapter on Treatment. The first question is as to the possibility of inducing absorption of this fatty histolytic material. Remembering that the lymphatics do absorb material no longer functionally useful, and that this is subjected to a secondary digestion, a fact insisted on in all works on physiology, it would seem consistent with this knowledge to prescribe iodide of potassium. But granting that the old matter is absorbed by this, and it is not even probable, is there any ground for supposing that in so doing any good is attained towards the endowment of the germinal matter with power to furnish competent histo-genesis?

Without this, any removal of the old material, even if possible, could only be futile, as regards any useful result. The association of the disease with the germinal spots in the early stages, and not with the portions of fibre more remote from them, points to its connection with defect in tissue production, rather than the non-removal of waste.

Whether any agent could be found which could affect this defective histo-genesis, or not, is problematical. The general rules of therapeutics for tissue-building, such as improvement in the condition of the blood, aiding in its depuration by arsenic, iodide of potassium, and eliminants generally; and the

improvement in it positively by the addition of iron, cod-liver oil, not inadmissible here (as it would be in obesitas cordis, or fatty infiltration), good, nutritious diet, with fresh air, and carriage exercise, may be of some service. In those cases of a vicious alcoholic diet too long persisted in, good results may fairly be expected to accrue, to a greater or less extent from this treatment, and it has its advocates; but it is in those cases chiefly that it can do any good, while there is room for suspicion that in many cases an error in diagnosis has been made, and that condition, which in its signs and symptoms simulates closely fatty degeneration, viz., an anœmic condition in the old or aged, alone exists, and is remedied by appropriate treatment. No such suspicion, however, could attach to the observations of Dr. Warburton Begbie who, in his lectures, used to speak favourably of this curative treatment by diet in many cases of fatty degeneration. But there is not much ground for hope in the direction of any treatment other than palliative.

The palliative treatment of structural diseases of the heart, whether existing alone, or allied to valvular lesions, and they are not incompatible by any means, especially in the more advanced stages of valvular lesions, is that from which most must be expected. And the first point to be insisted on is the avoidance of effort. If caught in a thunderstorm, away from shelter, the patient must prefer getting wet to the skin, with the inconvenience of going to bed till his clothes dry, if change is not at hand; if any accident occurs he must avoid all interference, the shock is quite deleterious enough for him; he must avoid bustle and crowds, must be in time for trains and omnibuses, &c., and must prefer to miss them to making efforts, which may be fatal. He must regulate his bowels, and not strain at stool; sexual intercourse is not free from danger, though the passion is too diminished to make it a source of probable danger; still, if found along with commencing locomotor artery, it is not a mere hypothetical source of danger. He must avoid mental excitement, politics, crowded meetings, where the atmosphere befouled with carbonic acid, might produce dangerous fainting; and blood laden with carbonic acid not only partially

anæsthetises the heart, but makes the blood flow less easily along the blood-vessels (Wm. Henderson, Edin.). All monetary speculations must be abandoned; the excitement is not free from danger, while the brain, imperfectly supplied with arterial blood, is liable to erroneous calculations, to imperfect thought, and is no longer suited to complicated mental processes.

The patient's family must also be apprised that the cerebral working is impaired, and petulance, perversity, puerile or childish mental obliquity must be attributed to the physical disease: indeed, more charity must be extended to the mental processes of the aged, who no doubt ought all to be Nestors, but are not, a condition not confined to the councillors of Job, but found even in more recent times.

The patient's whole life must assume the character of the invalid, and as the disease in the heart walls is not to be remedied, a general lowering, or "levelling down," must be insisted upon until a point is reached where the general manifestations are more on a par with the condition of the heart, and a species of equilibrium attained, a balancing of various parts in power, which is most favourable to the continuance of life. The preservative agency of lowered vitality is a most important matter to bear in mind in the treatment of incurable disease.*

This general maintenance of a quiet invalid's existence, in the more advanced stages, and of avoidance of effort and exertion in the less pronounced cases, may be somewhat aided by action on the fibres of the heart, remaining sound, by digitalis. Dr. Quain (Lum. Lect.) endorses my suggestion, made in Digitalis, to strengthen such fibres as remain by the administration of digitalis. By avoidance of all call upon the heart, by proper hygienic measures and attention to secretions, and with this more especial treatment, he says, "many cases will improve considerably." Considering Dr. Quain's attention to this disease, his reputation and his consequent extended experience, this statement is very encourag-

* A paper to this effect was read by the author at the Newcastle Annual Meeting of the British Medical Association in 1872, and produced a lively discussion, in which Professor Hughes Bennett and others took part.

ing. There is one encouragement, however, not intended by it, and that is a too great confidence in our remedial powers, for he also states, "In treatment we must bear in mind the cause of the affection," and divides the cases into those depending on a general state, and those where it is rather a local condition.

Of course where there is yielding of the ventricular walls, with venous congestion, &c., the administration of digitalis may be of service, but it is by its effect on the sound fibres which are yielding, and not by any action over the decayed portions. The dilatation, and such symptoms and consequences as arise from it, may be somewhat relieved by this agent, but it is obvious that such a condition is not favourable to the action of any remedy; and when dilatation, and its sequelæ, follow fatty degeneration, the comparative failure of digitalis, or anything else, can be no matter of surprise. The same must be said of all structural diseases of the heart, as compared to simple dilatation in a structurally healthy heart, and the apparent failure in the action of the remedy, which, of course, is not a panacea for all heart disease, may occasionally lie in the imperfection of the diagnosis, rather than in the agent.

The treatment of the attacks of angina pectoris is notoriously unsatisfactory, and diffusible stimulants of all kinds are administered. The addition of digitalis has appeared to the writer as useful in these cases; and where there is reason to surmise it due to temporary distension, this is explicable. But the variously described pathology of angina, for no more positive statement is yet warranted, makes all treatment empirical.

Brunton tried nitrate of amyl successfully, but Horatio Wood, M.D., in the Warren Prize,* failed to satisfy himself, by experiment, that we yet knew enough of nitrate of amyl to indicate its therapeutical usefulness. Allowing the patient to remain perfectly undisturbed, unless the position is positively injurious, is always advisable.

The other structural diseases of the heart are diseases connected with the muscular walls, but not with the muscular fibrillæ themselves directly. The muscular structure

* "American Monthly Journal of Medical Science," July, 1871.

suffers as a secondary affection as the result of compression, but there is no disease within the sarcolemma. The disease is essentially in the interstitial connective tissue, a cell growth in the intermuscular areolar tissue.

Fatty Infiltration.—Here there is, as shown in Rindfleisch's plate, a growth of fat in the intermuscular structure which becomes destructive by its pressure on the muscular fibrillæ. These are seen to be compressed, diminished, and ultimately destroyed. In proportion as this goes on the heart becomes weaker and less efficient, and the symptoms of organic heart disease call the patient's attention to his own condition. The deposit of fat, not uncommonly called obesitas cordis, is not always scattered through the structure to the heart, but may arise from the fat which normally occupies the sub-serous, or sub-peritoneal tissue, and covers the heart. When so occurring, it proceeds gradually inwards and advances along the connective tissue, compressing and destroying all within its reach, as is the wont of interstitial affections. In time the heart is found to consist of a mass of sub-serous fat of a definite thickness, so that where the muscular structure is thick a considerable amount yet remains, as in the left ventricle, while the right ventricle contains only a thin layer of muscular fibre immediately under the endocardium, with the columnæ carneæ and papillary muscles. At other times it is not so especially connected with the outer surface of the heart, but is more scattered.

The symptoms of this affection are those of impaired action of the heart without valve-failure. The usual symptoms of hurried respiration, feeble, faltering pulse, and diminished first sound are found, just as in fatty degeneration of the muscular fibrillæ. The diagnosis is not easy, but the points to remember are, that with this increase in fat comes increase in bulk and in percussion-dulness, while this development of fat accompanies general obesity, especially when it sets in in elderly persons. There is rather cardiac dyspnœa than angina pectoris, and the patient does not present those degenerative changes which accompany the tissue-necrosis. In the one there is evidence of good assimilation and deposit of fat; the other, though not necessarily

a lean person, gives not this impression, but rather that of impaired tissue-formation and repair.

Diagnosis.—In forming a diagnosis, there must be a sort of calculation of probabilities, guided by any chance light which may be thrown on the case by its own peculiarities. But the distinction between fatty degeneration of the muscular structure of the heart (Fettenartung) and fatty deposit or infiltration (Fettneubildung) is rather pathological than clinical. It is but proper, however, that they be considered separately, being, as they are, two such clearly distinct diseases. Many cases of fatty tissue-decay are too marked for any doubt, and in other cases the probability of fatty deposit is so strong as to leave small room for doubt. The difficulty would lie in cases of cardiac debility in persons of full habit, and with a marked development of fat. But it would not practically be of much importance to establish a diagnosis, as the lines of treatment would be the same.

Prognosis.—The difference would lie a good deal in the prognosis, which is better in fatty infiltration, as admitting, as a possibility, of curative measures. Still, where much of the muscular structure is destroyed, it is questionable how far it could be repaired by new growth.

Treatment.—Under either circumstances, supposing it were possible to diagnose them perfectly, the treatment would be directed to the development of nitrogenized tissue, and the removal of fat. The use of alkaline waters, Vichy, Seltzer, Carlsbad, Kissingen, Homburg, either on the spot or in bottle, or a visit to British alkaline springs may be of great use. There can exist no doubt that alkalies do tend to break down the accumulation of fat within the body. Small doses of iodide of potassium, along with fifteen or twenty grain doses of bicarbonate of potash, three or four times daily, may be used for patients who prefer it to the use of mineral waters. But each dose should be accompanied by a full draught of water after it: the dilution of alkalies has a great deal to do with their being effective, let the purpose for which they are administered be what it may. Also, no doubt, hydrocarbons should be taken but sparingly, and more especially in the concentrated forms of fats, oils, butter,

&c. Perhaps, however, sugar is as objectionable as anything, not only for its ready assimilation, but its tendency to aid in the assimilation of other hydrocarbons. The entire deprival, as far as is possible, of hydrocarbons and the substitution for them of an unlimited supply of nitrogenized food, commonly known as the system of *Banting*, is a dangerous liberty with man's construction, as not being entirely a carnivorous animal. The attempt to procure this sudden metamorphosis is about as likely to be successful as would be an attempt to procure an equally marked variation of type in support of Darwin's views. No doubt certain tribes have become from necessity almost entirely meat-eaters, and an hereditary transmission of the power to feed on it, implied by survivorship, may enable them to do so with comparative impunity; but it cannot be supposed that a medical consultation, or the perusal of a pamphlet, can effect this accommodation. The deprivation of hydrocarbons gives a craving for alcohol, let the explanation be what it may: and the Indian and the hunter do not abandon themselves to debauches from sheer depravity, and a course of Bantingism produces similar inclinations. Beer and porter are not only alcoholic, but are largely charged with sugar, called in brewer's phrase "saccharine," and are decidedly to be withdrawn from the patient with obesity. Food calculated to fill the stomach without enriching the blood, and so leading to its feeding itself on its stored-up material, as the parenchyma of vegetables, fish, in all its forms, &c., is desirable. In a little time the cravings of the appetite become much weaker, and the self-denial is more bearable, while the feeling of lightness and activity produced rewards and encourages the patient. Denial, or, in other words, hunger, is much to be preferred to an unlimited supply of azotized food, and a marked increase in the amount of chronic Bright's disease has unquestionably followed the recent fashionable attempt to metamorphose man and woman into a carnivorous animal. The azotized food is partially and imperfectly oxidised, and the excess of work thrown on the kidneys leads to disease in them.* Light and subacid wines should not be given with

* The analysis of urine after a non-azotized, a mixed, and a purely azotized

a highly nitrogenized diet, as they do check kidney action, and lead to disease in the kidneys, as the post-mortem table of Vienna abundantly testifies. Gin or whisky, Scotch or Irish, with potash or Seltzer water, are preferable, or a little sherry and water. What effect the Fucus vesiculosus, or common bladder-wrack, really has on the absorption of fat is yet undemonstrated: it was at one time in vogue as a cure for obesity. When once the effect of abstinence is marked by improvement, then moderate and increasing exercise may advantageously be added. The French advocate the withdrawal of fluids as a treatment of obesity.

False Hypertrophy.—Any increase in the heart's structure by anything other than true striped muscular fibre, could be designated "false hypertrophy." As, however, other diseases of the structure of the heart walls possess more marked pathological characters, it is desirable to restrict this term to an excessive development of connective tissue in the intermuscular areolar tissue. This occurs to such a length that the muscular fibrillæ become compressed and wither, as in fatty infiltration. From this come symptoms of heart failure, for there is nothing to lead us to suppose that the excessive connective tissue could give symptoms of its presence. At one time it was conjectured that hypertrophy of the heart was not merely due to increase in true striped muscular fibre, but that fibres of lower grade and imperfect function also occurred, as fusiform, unstriped, involuntary, muscular fibre, and even cell elements. We now know that true hypertrophy is due to hyperplasia of perfect fibres, and that in false hypertrophy there is increase of connective tissue merely. This may attain a great development, as in a heart in St. George's, weighing 40 oz., the increase being found to be connective tissue, and not muscular fibre.

Causation.—Various hypotheses of causation have been hazarded, the favourite one being that it is due to congestion of the coronary veins, founded on its pathological relationship to the development of connective tissue in the liver, spleen, and kidneys, in obstructed circulation from heart

diet, shows, by the increase in urine salts, the increase in work the kidneys necessitated by an azotized diet.

failure. Probably these other conditions are usually found along with it, but there is a difficulty about the affection of the heart itself. Is it the consequence of obstructed blood flow? Or is it the cause? May not the other similar conditions be due to its action on the heart, and not it a result of venous congestion. It is not by any means clear; and the presence of an advanced condition of development of connective tissue does not settle anything; for Sir William Jenner has pointed out that the heart walls so affected do not collapse on section, restraining their shape, and recovering it when bent by their own elasticity, like an india-rubber ball. Now this condition of the heart walls is not by any means so common as a development of connective tissue in other viscera, a fact militating seriously against this view of its origin. Another important fact is that the coronary veins open through the walls of the right auricle obliquely, like the ureters into the bladder, and thus not only are the edges of the openings closed by distension, but the muscular fibres, such as there are, and they are increased in distension usually, being stretched, would oppose reflux by closing the vein betwixt them. The coronary circulation is especially provided against the effects of distension reaching the heart itself.

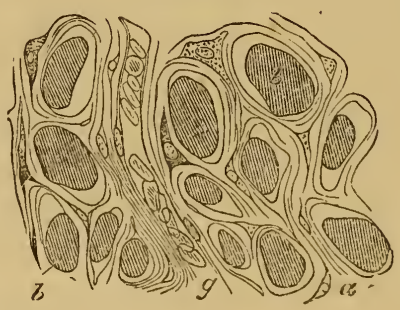
Another view is this, that this is an interstitial inflammatory product, the result of interstitial carditis, and that it is to be met by the administration of antiphlogistics, and other anti-inflammatory remedies. The first difficulty in the way of the treatment would be the difficulty in diagnosing the condition, until its effects on the muscular fibre were apparent in cardiac debility; and nextly, the slight action exercised over these interstitial inflammations by any remedies yet known to us. The *first* pathological hypothesis is the more probable of the two, though its acceptance is not nearly so general.

It is to be feared that too little is yet known about this affection to warrant any remarks as to its symptoms or prognosis. Consequently it is equally futile to make any remarks on the treatment of a condition which cannot be recognised and distinguished in life from the other interstitial affections

of the heart which increase its bulk. This affection must still be considered as a *post mortem* table phenomenon, and nothing more.

Atrophy.—Atrophy of the heart is a disease which does not mean merely diminution of bulk, though that is the most marked form of it, but merely a reduction in the muscular fibre. As simple atrophy or reduction in size, it is found in the course of wasting diseases, as phthisis, cancer, &c. It is then termed simple atrophy. When the term excentric atrophy is used, the condition of simple dilatation is indicated, which is a much preferable term in every way. This condition of enlargement of the ventricular cavities, and stretching and thinning of the muscular fibre, from its being accompanied by no increase in the amount of muscle, has been described as simple dilatation in a previous chapter, and need not be again referred to. Rindfleisch describes, as an atrophy of the heart, muscular wasting with development of connective tissue, and thus no diminution of bulk, well illustrated by the accompanying plate:—

Fig. IX.



False Hypertrophy—
a. Excessive Connective Tissue.
b. Compressed Muscular Fibre.
g. Capillary full of Blood.

He calls this development of connective tissue compensatory to the wasting of the muscular-primitive bundles. It would be just as possible, and perhaps more probable to describe it as an increase in the interstitial connective-tissue, and wasting of the muscular tissue from compression. Of course this definition of it and the first one have been fought over again and again, in reference to the development of this

condition in other viscera. It can only be settled by a reference to the behaviour of connective-tissue in other parts, as in the shrivelling of valves, with patches of parenchymatous connective tissue in them, the behaviour of it in cicatrices, &c. It shows a marked tendency to contract and produce mischief thereby, where no prior atrophy is supposed even to be present. It is surely, then, odd that it should be found in excess, along with wasting of normal parts, so singularly associated with this wasting, too, and yet be innocent causatively. On the other hand wasting may exist without it, so that the presence of this excessive development of connective tissue, in atrophy of normal tissues, must be regarded with some suspicion as the cause of that wasting.

Causes of Atrophy.—These are general marasmus, and a partaking in a general condition, pressure on the heart by pericardial effusion, and partially arrested blood supply. It may, however, exist congenitally, and very small hearts have been found in comparatively grown up persons. L. Beale, in his work on the urinary organs, speaks of a class of persons with small hearts congenitally, small bones, and general plumpness, who seem to give way to kidney disease, and general breaking up at an early age in a strange manner.

Symptoms.—There are the usual symptoms of cardiac debility, with increased rapidity. Niemeyer says that simple atrophy produces a more marked effect on the pulse than when it is accompanied by dilatation. The heart's impulse is imperceptible, and its sounds thin and feeble.

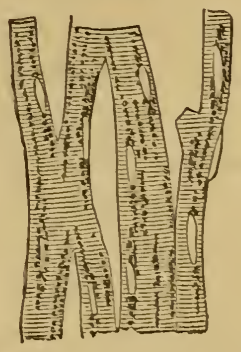
Prognosis and Termination.—The termination is untoward, except in the form due to pericardial effusion and pressure, and the prognosis is in accordance with its termination and its pathology.

Treatment is out of question except in the relief of pericardial effusion, when that is its cause.

Brown Atrophy.—This is an affection found in the hearts of the aged, and first described by Rokitansky. It consists of a development of pigment within the primitive bundles, and extends through the whole heart. Whether it is derived from the colouring matter of the blood, or muscles, is not known. It has no symptoms, is unknown clinically,

and its interest is purely pathological. A good idea of it will be formed from the following plate from Rindfleisch, who states that it is found in marasmatic conditions, not necessarily confined to the aged:—

FIG. X.



Brown Atrophy (after Rindfleisch).

Amyloid Degeneration.—This is a somewhat rare affection of the heart walls, and consists, according to Oppolzer, of the formation of a shining, colloid-looking material within the primitive-bundles. It gives the usual reaction of amyloid matter to iodine and sulphuric acid, which is not a true blue. According to Dickenson (on Albuminuria), this so-called amyloid material is really de-alkalised fibrin from long suppuration. It is usually found in the right ventricle, according to Rokitansky, especially when the right ventricle is enlarged.

It has no special symptoms, but is usually found along with enlarged amyloid liver, spleen of kidneys, with albuminuria and tube casts. I regret that it has been impossible to procure a plate of this disease, as it appears under the microscope.

Syphilitic Gummata.—These are not uncommonly found in the heart's structure in the subjects of constitutional syphilis. It has been termed syphilitic myocarditis, but the action, which results in these gummata, is not within the muscular-primitive-bundles, but in the interstitial connective tissue. According to Virchow, the products of these syphilitic interstitial inflammations in muscles is a growth of fine granulation corpuscles, which not uncommonly undergo

fatty degeneration into cheesy masses, at one time mistaken for tubercles, not miliary, but the masses once known as the yellow tuberculosis of Rokitansky. At other times these gummata become converted into fibrous masses of connective tissue.

The masses are scattered through the substance of the heart just as are gummata in other muscles, and are found from the size of a bean to a pigeon's-egg (Virchow), or a billiard-ball (S. Wilks). They are often accompanied by a thickened condition of the endo- and pericardium, and may be seen showing through the columnæ corneæ, under the thickened endocardium.

The largest are found in the septum ventriculorum (von Dusch), and project from thence into each ventricular chamber. They have been described by various writers, as Ricord, &c., and formed the subject of a notable discussion between Virchow and von Barensprung in the Deutsche Klinik, when Virchow demonstrated that they were not tubercular masses.

Symptoms.—There are the usual symptoms of cardiac failure, and as the disease occurs during the prime of life mostly, it is somewhat unaccountable. The diagnosis is formed on the absence of rheumatic fever, or valvular disease, absence of anœmia, or other debilitating cause, or atheroma, or general decay, &c., the positive evidence of constitutional symptoms, and possibly of gummata elsewhere.

Treatment.—This is the treatment of syphilis, and Lancereaux found the symptoms relieved by iodide of potassium. But the treatment must be resorted to early on, as no treatment can be of service, when once caseous degeneration has gone on, or when the masses are converted into fibrous masses of connective tissue.

Tubercle.—The true miliary, or gray tuberculosis of Larnnec, is also found in the heart walls, in the connective tissue, in cases of acute tuberculosis.

Cancer.—Cancer has been found to attack the heart, as carcinoma, medullary or melanotic cancer (Virchow). It is commonly secondary, but may be primary, and Bodenheimer has described a case of primary nodular cancer of the right side of the heart.

Parasites.—The heart is liable to be the seat of parasites, or entozoa, the cysticercus, cellulosa and echinococcus. They have been known to lead to rupture, and so cause death.

Polypi.—Polypus of the heart is usually merely a fibrinous mass, consisting of blood fibrin and white corpuscles, entangled among the chordæ tendinæ, or forming a mould of the conus arteriosus, and extending up to the artery, with the marks of the valves clearly to be seen on it.

They are usually found in the pulmonary artery, and are formed in the death agony.* But real polypi have been found growing from the inner surface of the heart. Grisolle collected authentic cases, and Flint (Diseases of the Heart, p. 287) describes a case which occurred in Bellevue Hospital, New York. In this case there were two polypi, both growing from the right auricle, and one extending into the right ventricle. The right side of the heart was much dilated.

The tumours were examined microscopically, and found to consist of, "1st, blood-vessels, some of which contained blood. 2nd. Cells, spindle-shaped, star-shaped, round, and oval. ·018 mm. in diameter, most of them being somewhat fatty. 3rd. Fibres in broad bands. 4th. Basement membrane, transparent, homogeneous, and finely granular."

There is no diagnosis of them in life given; but false polypi have been diagnosed in life, once by Dr. R. Drummond, in York Hospital.

* Various suggestions have been thrown out as to the prevention of the formation of these polypi, on the supposition that the polypi cause death. Bouilland thought they might be dissolved by the administration of agents internally. Richardson has suggested their prevention by giving ammonia. Legroux recommends potash and soda salts. My friend, Dr. Henry Barnes, of Carlisle, has recently been investigating the subject of fibrinous polypi, and informs me that in a case where ammonia was given to prevent the formation, which was apprehended, of this blood-clot, no good resulted, and the clot was found on post-mortem. From experiments with blood-clot, Dr. Barnes has found that no result follows from its immersion in a solution of carbonate of ammonia or of bicarbonate of potash; little effect from immersion in solution of iodide of potassium, and ready solution in lime, either in the saccharated solution (best) or lime-water (less effective). It is an interesting subject, but it is not so much the solution or prevention of the polypi, but the removal of the condition which favours their formation which we must hope for therapeutically.

CHAPTER IX.

RUPTURE OF THE HEART—TRAUMATIC INJURIES—DISPLACEMENTS.

Rupture of the Heart.—This occurs usually in hearts previously affected by either fatty degeneration, abscess, or parasite, as hydatid cyst, or is induced by a blow, or fall, or other injury. It is a not uncommon termination to fatty degeneration. The seat of the rupture is given by Quain as most commonly in the left ventricle (76), (and Rokitansky points out that it so occurs usually through the apex, which is most liable to localized degeneration,) then the right ventricle (13), the right auricle (7), septum (4), and left auricle last (2). The rupture is sometimes single, and in other cases several tears are to be detected, some with a clot of blood in them, as if repair was being attempted, even under such unfavourable circumstances as the heart's unceasing labour. It would appear from this that a partial tear may occur, and possibly a rupture is only complete some time after the tear was commenced. This bears on the causation, as mentioned before, and often an apparent unprovoked rupture really took its origin in some unobserved effort made some little time before. The tear is often jagged, and not direct, as if one layer of fibres had given way after another, near, but not at the same spot, the seat of the rent in the first layer. These ruptures are usually regarded as occurring from within outwards, but my friend Dr. Alfred Wiltshire informed me of a case where the rupture was from without inwards, and imperfect slits were found on the outer surface of the heart. This has also been found by others. The question of whether rupture takes place commonly from within outwards, or from without inwards, is of interest, as bearing on the question of its production during systole or

diastole; and also whether it was produced in systole by internal pressure, or from the outside fibres tearing themselves in an attempt to compel ventricular contraction. When the rupture is from without inward, it would favour the supposition of the rupture being due to the last cause. When from within outwards, it is possible that it occurred either from ordinary distension during diastole, or from increased centrifugal pressure during systole, on a diseased and non-acting part, which would be in proportion to the centripetal pressure exercised by the acting sound portions of the ventricle. This last would seem corroborated by its occurrence in the left ventricle most frequently, next in the right, &c., while its occurrence on the right auricle more frequently than the left would suggest the hypothesis that the rupture occurred in diastole, the right auricle being thinner, and subjected to the influx from the systemic circulation, and the left auricle being thicker, and subjected to influx from the lesser or pulmonic circulation. Before general distension a weak part would most readily give way wherever situated, but the probability of the rupture being caused by centripetal pressure during systole is borne out by the circumstances under which it occurs.

Immediate Causes.—Though rupture has occurred during sleep, there is reason to suppose that the completion of it only took place then. It occurs during sudden call on the heart by mental agitation or alarm, by bodily exertion, dancing, springing, prolonged marches, coition, effort at stool, epileptic attacks, or arrest of the peripheral circulation in a cold bath (von Dusch). This would favour the hypothesis of systolic rupture from increased ventricular action and thus increased general centripetal pressure on the fluid contents of the ventricular chamber, and increased centrifugal pressure on the diseased and non-acting parts.

Symptoms.—The symptoms are those of collapse, where instant death does not occur. The skin is cold; the pulse small and irregular, and almost imperceptible; and, indeed, the same appearances as shock, or fainting, without necessarily loss of consciousness. The diagnosis is rather of probabilities than certainties, but if there is also increased

lateral dulness, from the pericardium being distended by blood poured into it, the condition is more surely indicated. The sudden occurrence of this accident, the sudden shock to the patient, the intense cardiac suffering, breathlessness, and feeble, irregular pulse, with loss of heart sounds, and the collapse continuing, aid to indicate the diagnosis.

Prognosis and Terminations.—The prognosis is that of death, certain and unavoidable. It, however, may not occur for some time, even so far as six or eight days. The rupture is partially closed by a clot when the fibrous pericardium becomes so distended as to oppose any further flow of blood into it at each systole.

Treatment.—This must depend upon circumstances, and must necessarily be merely palliative, and not curative, in any sense. It is a complex question how far stimulants do not increase the flow through the rupture; but if they give the patient ease, it is something. Narcotics may be indicated.

Traumatic Rupture.—The heart, in common with other viscera, is liable to be ruptured by external violence, as falls from a height, being thrown violently by machinery against some resisting body, by compression, as by rail-carriage buffers, &c. The rupture here is through the thinner and less resistent parts of the heart, as the right ventricle and the auricles, the left ventricle not being affected nearly so often. This is the reverse of rupture from disease. Death inevitably is the result, unless the septum be ruptured, and then cyanosis results.

Wounds of the Heart.—The heart is liable to be wounded by penetrating wounds of the chest, whether bullet wounds, sabre, bayonet, or knife wounds, either self-inflicted, homicidal, or in war; it has also been unintentionally wounded in excision of the mamma and other tumours over the left breast, and has been pierced with a trocar, when a dilated heart was mistaken for a collection of fluid in the pericardium. It has been wounded by needles and pins, &c., fixed in the œsophagus; in that case the posterior surface of the left ventricle was injured. From wounds inflicted in front, the right ventricle, from its position, is most commonly wounded, but the right auricle and left apex are also exposed.

The heart has been partially cleft, a sword cut in the septum ventriculorum, with what result cannot be remembered. Many strange facts are recorded in connection with injuries to the heart, but these belong rather to collectors of teratological data than to a sober treatise. Bullets have been found encapsuled in the substance of the heart, and without any external scar, giving grounds for the hypothesis that they must have penetrated one of the great trunks, and fallen into the ventricle.

Wounds of the heart are usually fatal, but it would not appear necessarily so, unless they are penetrating, or cut a coronary artery, or one of the larger branches. Pericarditis is, no doubt, a result of injuries through it; but the whole subject is too unfrequent to give as data as to what occurs after wounds of the heart--not being immediately fatal.

Treatment.--Where the wound is slight and not through the heart walls, the patient must be kept quiet in bed, and if the heart's action is tumultuous, aconite might be given. Local treatment is out of question.

Displacements of the Heart.—The heart may be displaced by a fall, or other violence, by disease, or may be congenitally out of its ordinary place. The subject is of interest as bearing on the diagnosis. So accustomed are we to think of the heart being on the left side, and associated with the left nipple, that when not so situated some doubt might exist as to the diagnosis. We are now pretty familiar with the fact that the heart is commonly displaced to the right by effusion into the left pleura, and from the formation of pleuritic adhesions, &c., it may remain there. It is also liable to be displaced by an emphysematous lung, or drawn over by retraction and shrinking of the right lung, by cancer, and by diaphragmatic hernia (Da Costa). A case is related by Cheyne, of Dublin, in the Cyclopædia of Practical Medicine, of displacement of the heart to the right side, occasioned by an accident, where it remained, palpitating a good deal till it got accustomed to its new quarters. Here simple rupture of the pericardium must have allowed the heart to escape, while it did not admit of its being drawn back by the elasticity of the vessels.

Stokes had once a case of disease in a person in whom the heart was congenitally on the right side, and diagnosed it correctly.* The positive presence of the signs of disease of the heart in the right half of the thorax does not invalidate the diagnosis, only, of course, such cases are rare. The Pathological Museum of Vienna, however, now possesses two specimens of reversal of the position of the viscera, the heart and spleen being to the right side and the liver to the left. My friend Dr. Elliott, of Carlisle, has had long under care a patient in whom he has diagnosed this condition, and his opinion is corroborated by Sir Robert Christison and others.

* "Diseases of the Heart," p. 463.

CHAPTER X.

General Treatment of Heart Disease.

The treatment of diseases of the heart is a subject on which unfortunately too much doubt and too little hopefulness exist. While it is perfectly true that a diseased valve can never be restored, and that the molecular necrosis, fatty degeneration, is not a very good subject on which to demonstrate the efficacy of remedies, it must not be taken for granted that all disorders and diseased conditions of the heart are equally removed from the reach of therapeutics. In a great number of cases of mere failure of the muscular structure of the heart and dilatation, not complicated by valvular disease, restorative treatment is followed by most satisfactory results. Such cases may occur after debilitating illnesses, terminating in protracted convalescence; after long and arduous exertion, as in nursing the sick, where one person is too unequal to the necessities, and where the slight rest permitted is too often taken in the sitting, and not the recumbent, posture; long over-exertion in agricultural service, or in large works, excessive gymnastic exercise, &c. Here the removal of all call for exertion, perfect rest, especially in bed for a time, and proper remedies, will give great relief in every case, and in a large number perfect restoration to health is permissible. Even when a valve is injured, great compensatory growth of the muscular walls will permit not only of fair health being maintained, but even of great exertion being undertaken, without any bad results.

Niemeyer relates a case of a huntsman of the Griefswald who, though suffering from aortic regurgitation, went through forced marches, and other severe calls on the powers, in the war of 1866 quite successfully. Growth in the left ventricle compensates more or less perfectly for disease at the aortic orifice; in stenosis the increased power enables an equal amount of blood to be driven through a smaller opening in

an equal space of time, and thus, for long, of perfect compensation being maintained; and the above case of Niemeyer's shows how the hypertrophy, so invariably found to a marked extent in aortic regurgitation, had resisted the backward flow through the incompetent valves on the aortic recoil.

Mitral disease is met by increased growth and power in the right ventricle, which is not lost in the short pulmonic circulation, and stenosis and regurgitation are thus met, as by the left ventricle in aortic disease. Thus mitral disease will continue for years compatibly with a fair amount of bodily vigour, and without much inconvenience to the sufferer. I have seen growth, and rapid growth too, with the perfect establishment of puberty, well maintained in a girl with a very considerable mitral regurgitation, and whose condition at the commencement of treatment was far from encouraging. While we know only too painfully well that a distorted or contracted valve is beyond the reach of our art, and that though connective tissue is soluble in a solution of barytes, we are yet as far as ever from seeing how to remove the growth of it, which has injured the valve, even if it were possible to hope that after its removal the valve would assume its former and normal shape. Still this muscular compensation, so successfully established by the self-curative power of the system itself in many cases, is to some extent under our control. In all systems of good reparative power all call upon the muscular walls leading to distension of the ventricular cavity, as we saw in Chapter VII, tends to produce hypertrophy.

This, there is reason to believe, is thus brought about. Distension which involves the elasticity of the muscular fibre itself, that is, extending beyond normal distension till the full length of the fibres is reached, and involving the secondary elongation of the fibres, or their elasticity, creates an impression on the cardiac ganglia. This is quite in accordance with what we know; it is merely the normal stimulus in excess. . From recent experiments we learn that the heart possesses a sensory nerve, a fact which we could conjecture from the pain accompanying heart affections, and especially acute ones, and this nerve has

been found to be a vaso-inhibitory nerve, *i.e.*, it arrests vaso-motor nerve action and dilates the peripheral muscular extremity of the circulation, the arterioles. Thus is not only tension of the arterial system lessened, and with it the opposition offered to the heart's more completely emptying itself at each systole, but this affects the arterioles of the coronary circulation, and thus "a broader and more rapid stream of blood" is furnished to the heart itself; and so leads to hypertrophy. This is the causation of hypertrophy as it appears in the present state of science. It is borne out not only by the causes of hypertrophy, mentioned in a previous chapter, but also with Traube's famous definition of hypertrophy, without visible dilatation, or complete compensation; hypertrophy with dilatation, or incomplete compensation; and simple dilatation, or no compensation. Distension leads to dilatation in systems unequal to compensation, and is arrested by hypertrophy ere dilatation is established in systems with better vital force; while all grades of intermediate, incomplete, compensation are found. This is nature's treatment, but it is not always successful. Much, however, can be done by therapeutics. The blood itself may be enriched, and the dilatation may be due to anœmia or spanœmia, by hæmatics, iron, &c. While agents which increase ventricular contraction not only arrest the dilating process, and tend to institute a return to the normal size, but also affect the circulation most advantageously. For we know that dilatation takes its origin in incomplete systole, and a certain amount of blood remaining in the ventricular chamber, when the blood rushes in from the veins; for the blood will pour into the ventricle as long as the pressure on the ventricular wall is less than on the venous walls, *i.e.*, until the pressure in veins and ventricles are equal, and no further. But when the ventricle is imperfectly emptied, a compromise takes place, in distension of the ventricle and engorgement of the veins, in exact proportion to the amount of blood already on the ventricular chambers. This is self-evident enough; it is a mere question of physics.

Thus, with dilatation we get venous engorgement, one source of distress and mischief, and imperfect filling of the

arteries, another source of trouble. For the elastic arteries transmit what is poured into them. If, then, a small bulk of blood only is thrown into them at each ventricular systole, the supply of arterial blood to all parts is diminished, including the heart itself. Thus dilatation has a tendency to perpetuate itself, by this imperfect blood supply, in spite of the beautiful nerve arrangement for increased nutrition in distension.

But when an agent increasing ventricular contraction is administered, more perfect ventricular contraction passes, at each systole, a larger bulk of blood into the arteries, this leads to better supply of arterial blood generally, and relief of all parts. But this includes the heart itself, thus: 1. More perfect ventricular contraction causes a larger bulk of blood to be thrown into the arteries at each systole. 2. This produces more perfect arterial distension. 3. This, in the elastic arteries, leads to more perfect recoil. 4. The aortic systole is the propelling power into the coronary circulation, and more perfect recoil leads to improved coronary circulation; and 5. This conduces to more perfect nutrition of the heart itself.

But, at the same time, another important matter has been brought about. We saw in Chapter I that increased rapidity in the ventricular contractions took so much off the heart's sleep, *i.e.*, so many more contractions in each minute took off the time, taken up by these abnormal contractions, from the diastole, or period of rest. This distension of the heart chambers induces rapid imperfect contractions; and more perfect contractions allow of a longer diastole. The effect of slowing the pulse is universally allowed to digitalis, when that pulse is abnormally fast.

Thus we have secured a longer diastole and so many fewer systoles each minute, or longer sleep, at the same time that we have improved the blood supply to the heart itself. This is all sober fact, but its effect in aiding a heart, not quite equal to hypertrophy by its own efforts, is often wonderful. These positive curative measures of rest, diminished call on the heart for exertion, improved condition of blood by hæmatics, and direct action on the heart by agents which

induce ventricular contraction, will often establish a hypertrophy, if the dilatation is irremediable; at other times restoration of the heart to the normal; and even when a valve is positively injured, a great approach to this can be attained.

In a great, a very great number of heart cases, hopelessness is unfair to the patient and unworthy of our present knowledge. If in some cases where molecular necrosis is slowly cutting down the muscular structure, we must look on with a feeling of incapacity to be of much service; it is not so in a large majority of cases. Both in justice to the patient and ourselves, hope can be held out, and despair need not be indulged in. "We see too much of the recovery of heart power by rest, food, and proper medicines; we know too many pulses that are beating happily now that we knew to intermit or be irregular years ago" (*Lancet*, leader, April 27th, 1872), writes a well-known medical authority, in speaking of the treatment of diseases of the heart, as it can be conducted now, to permit despair. The fact that a great proportion of cases of disease of the heart comes now under the heading of curable affections, and that another large proportion can be relieved, when not admitting of cure, is as certainly ascertained as that other forms are incurable and admit only of palliative treatment, and that, too, to a limited extent. Which form he has got before him, must usually be determined by the diagnostic skill of the practitioner, and on that diagnosis lies very often the practitioner's hopefulness, and with it the patient's chances of existence. For if time be lost, the case may proceed to a point admitting to a much smaller extent of treatment than when medical advice was first sought. There is a grave moral importance, involving one's duty towards one's neighbour, a very elementary moral law, about the formation of a diagnosis very often in medicine; for on that diagnosis often hangs the prognosis and the treatment. And treatment may often much affect the prognosis, and no treatment, or improper treatment, may verify a prognosis in a very unpleasant manner. Even when in doubt, it is better to err on the side of treatment, which is not harmful even where it is not successful, and may often achieve results not hoped for, than to allow valuable time to slip

away, and a fellow-creature's life to dwindle out for lack of aid ere it is too late.

It is but a base return for a patient's confidence to meet it by an ignorance which is removable, or a hopeless scepticism which is not justified by facts. Had I not too often seen what is now being described, perhaps the expressions might be somewhat moderated, but they are within and not over the mark.

One of the most unfortunate scepticisms in relation to diseases of the heart arises from the often wonderful effects of rest. This is mostly seen in hospitals. A patient with dyspnœa, cyanosis, feeble irregular pulse, and small bulk of urine passed in the twenty-four hours, is taken in and put into bed by the house surgeon, or clinical clerks, in the presence of a class of students. No treatment is decided upon until the case is seen by a physician, and in the mean time the patient improves very much, his cough is less troublesome, his respiration less impeded, he can breathe lying down, or with much less propping, and the pulse becomes much steadier. This improvement is so striking, that "the expectant treatment" is continued until the patient is sufficiently recovered to resume his occupation. The case creates comment, and illustrates beautifully the effect of rest as part of the treatment of heart disease, leaving the impression on many minds that rest in bed is the treatment, *par excellence*, for heart failure. But follow that patient a little longer, and see the effect of the resumption of exertion, the toil necessary to procure the daily bread, and you would see every symptom return with its former or increased force. The heart is quite equal to the call upon it when lying in bed, and even to somewhat of compensatory growth under these favourable circumstances, but the resumption of toil soon brings out its incapacity. Rest in bed, valuable as it is, is only part of the treatment of heart failure, and will do little for those who must work or hunger, except as an occasional indulgence; and as such it is most valuable to many of our toiling populations, especially in towns where they can be received for a few weeks into a hospital ward.

For the more perfect general comprehension of the subject of the treatment of heart disease generally, *i.e.*, the elements of treatment, to be applied to each individual case according to its necessities, by the good sense and at the discretion of the medical attendant, the treatment will be divided into three sections: 1. The removal of causation, as far as is practicable; 2. The means of acting upon the heart itself; and 3. The rational treatment of sequelæ, *i.e.*, the palliative treatment of the consequences (rückwirkung) of heart failure.

Removal of Causation.—In the treatment of all affections, this constitutes the first step; but it is unfortunately too often beyond our reach. In the heart, however, which acts to a large extent mechanically, this is possible to a great extent.

All call for exertion soon tells on a heart which no longer possesses any spare power, but is actually unequal to its labour, to the demands on it. This effect of exertion upon the heart has all along been used as the test of structural inability, as the means of diagnosis betwixt the objective symptoms which are alike the result of disordered innervation or of structural weakness. It becomes of great importance then that when cardiac symptoms show themselves in the hammerman or striker, the collier, or other person, whose occupation necessitates violent efforts, to insist upon some other form of occupation making less demand upon the heart. This is often a difficulty to a working man, but the alternative must be put before him. For it is only too obvious that the form of labour which evokes the failure must be abandoned; for the causes which could induce disease in the healthy heart must have a sadly deleterious action when that organ is commencing to yield, and the various forms of compensation to become imperfect. No measures, however successful in affording relief to symptoms, can be expected to endow the organ with the virtue to resist the stress with impunity. If, then, the labour is persisted in, an unfortunate result can alone be anticipated.

The same may be said about exciting mental pursuits, and more especially in monetary speculators, and others whose occupations are exciting; for Quain, Richardson, and others

think, not unwisely, that the increased rate at which we live "in this madly striving age," exercises a decided influence in the production of heart disease, and that its increase in the death returns is not solely due to a better recognition of it, but to some extent to an actual increase in frequency.

All conditions which tend to weaken the patient, from bodily causes, must be removed, or relieved as far as it is possible. Thus indigestion, mal-assimilation of food, as preventing the proper nutrition of the blood, all debilitating discharges as chronic diarrhœa (this is in early stages; in latter stages it is somewhat different), dysentery, hæmorrhoids, bronchorhœa, &c., must be amended by treatment as far as they permit.

Imperfect blood-nutrition, however produced, leads to loss of propulsive power in the heart. Anœmia is ever to be averted in cases of heart failure. Flint (Diseases of the Heart, 1870) says, "The combination of anœmia and enlargement of the heart is to be prevented, if possible, and, if it exist, anœmia, if possible, is to be removed by appropriate measures of medication, diet, and regimen. Irrespective of this condition of the blood, all agencies which tend to weaken unduly the force of the ventricular contractions, are contra-indicated. In proportion to the weakness of the heart will be the tendency to dilatation rather than to hypertrophy. So long as hypertrophy predominates, the patient is comparatively safe" (p. 77). What gives this decided expression even more force is that here he is speaking of the treatment of hypertrophy.

When dilatation is manifesting itself, it becomes doubly important to avoid any tendency to anœmia; and where this tendency does not yield to treatment, but manifests an inclination to persist, the patient should be remorselessly confined to bed for a time, to reduce to a minimum the calls on the nutritive processes. This is often necessary after debilitating diseases, or where the heart itself has been affected, as in some febrile or inflammatory conditions, when the weakened muscular fibre is unequal to complete ventricular contraction upon its contents, and where both ventricular and venous distension are resulting.

Pretty much the same effect upon the heart as is produced by anœmia, is produced by nervous exhaustion of the ganglionic or organic system chiefly. This is what is meant by vital exhaustion. It is not mere mental tire, it is not cerebro-spinal exhaustion, but sympathetic. The effect of this exhaustion is to impair the sensitiveness of the cardiac ganglia, upon which depends cardia contraction.

When, then, from any impairment of this sensibility a condition of over-extension results, there follows imperfect contraction, and thus dilatation is inaugurated. All causes then which lead to loss of power in the pulse, and especially when this is accompanied by increased rapidity, indicating ventricular over-distension, should be avoided. These are excitement of all kinds, including excessive drinking on the one hand, and attendance on special lectures, meetings, or services on the other, the denial of sleep, especially in the horizontal posture, tobacco smoking to excess, similar use of tea, &c. Frequently it will be found that a case has such an exciting cause, so removed from common experience, as not to be found in any text-book, but which will at once point to its action and the necessity from its non-continuance.

The effects of all causes, or agents, which lead to nervous, vital, exhaustion, and increased frequency, with loss of power of heart contraction, is strictly analogous in effect, to imperfect nutrition or anœmia. The complete or partial removal, where complete removal is not feasible, of these causes is imperatively demanded in the treatment of the heart failure which results from them.

Where certain blood poisons, as in Bright's disease (see Chapter XIII), malaria, syphilis, lead-poisoning, &c., are the cause of impaired blood formation and nutrition, their removal by their various treatment, by specifics, so called, or otherwise, is clearly indicated.

A great number of cases of heart disease presenting themselves to the practitioner, whether simple muscular failure or accompanied by valvular disease, will be found with a fair amount of capacity for exertion, or in other words, in the early stage of the case. Here it is commonly only necessary to economise the forces, by diminished exertion, to avoid ex-

hausting causes, of all and every kind, and to resort to some of the combinations of remedies, mentioned in the next section, to cure absolutely in some cases, and in others to relieve for long your patient. With how much judgment and skill this is done will seriously affect your patient's chance of prolonged existence. It must also be borne in mind that relief, amounting to cure practically, will often be followed by forgetfulness of the physician's warnings, or a feeling that he might be too serious in his admonitions, or take too grave a view of the case, and it is ever easy to believe what we wish to believe, and the improvement in bodily vigour is followed by further calls upon it, the old groove is easy again to slip into, and a repetition of the causes will restore the condition of the heart, and again necessitate consultation of the physician; each time with a worse prognosis it will be found.

This was well seen in an old Irishwoman, a whilome patient, who was so quickly relieved by treatment, that she became quite indifferent to her dropsy and dyspnœa, getting rid of them almost so soon as she wished to. Relief was immediately followed by the resumption of a black pipe and the "cratur." The whole thing became very absurd, but in a few months it became serious, and too long delay in resorting to treatment was followed by rapid aggravation of all the symptoms, and death.

But unquestionably the tendency of most cases is to become steadily worse, often slowly and very gradually. Thus the patient relieved by treatment for a time, and the effect upon the heart of digitalis and iron persists for some time after their withdrawal, comes under treatment again, with either some consequential ailment or his symptoms brought back by some affection caught casually, but partly the consequence of his lessened power of resistance. This period of ill-health, or disorder, has brought out the heart's inability.

Treatment will probably restore him, if the disease has not been too serious, for some time, but it is only for a time.

The increasing dyspnœa or some visceral complication, the result of abdominal plethora, is now the complaint. Each time a more involved affection will call for a more elaborate

treatment, and simple action on the heart itself must be aided by some agent belonging to the last section, or the treatment of sequelæ. Again, a fair amount of health may be fortunately attained, but the case is gradually coming to a close, if not abruptly cut off by some sudden failure or pulmonary congestion. And of course the liability to sudden death increases as a condition of greater heart debility is reached, but it must be borne in mind the majority, and large majority too, do not die suddenly; indeed it is in one sense a pity that they don't die suddenly, somewhat oftener. The distinct tendency is to grow slowly worse till the defect in the circulation is affecting every organ, and felt in every function. In time the patient becomes almost a constant invalid, his permanent treatment of digitalis, iron, and usually some acid, or with a vegetable tonic containing tannin for the catarrhal stomach, supposited for the iron, only varied by some cough mixture, or by cathartics, and the use of the bath, or the application of some external treatment. The general principles of this treatment are arranged systematically, and the reader is thus reminded of certain remedial measures which his own intelligence will apply to the needs of each individual case, a combination often being indicated.

Means of Acting on the Heart itself.—The means by which we may act upon the heart itself are necessarily of three kinds—those acting on it from without, *i.e.*, affecting it regionally; those acting on it from within, the reduction of the bulk of blood circulating in it; and those which act upon its muscular walls directly. The action on the heart by pressure around it is only too obvious. This is most especially seen in the accumulation of fluid in the pericardium. But equally and much more commonly visible is the effect of pressure upon the abdominal surface of the diaphragm, and its bulging into the thorax, thus diminishing the space within which the heart moves in its action. The heart varies in bulk in systole and diastole, betwixt full distension and the chambers when emptied. It is obvious, then, that while the bony parietes of the thorax remain in *statu quo*, and the diaphragm is pushed upwards in the neighbourhood of the heart, the heart itself must be affected by that pressure.

That it is so is a fact well known in clinical experience, even in healthy hearts, as in accumulations of gas, solids, &c., in the stomach and colon: how much more they affect a heart already impaired is equally well known. The sense of discomfort, shortness of breath, and palpitation, which are found very unpleasant in a sound person, is intensified in a person with a diseased heart; and this more especially when the right ventricle is involved. The accumulation within the colon or stomach of gas must be met by carminatives, stimulants, diffusible or other; and in favour of ammonia has been urged a chemical argument. It has been asserted that a reduction of the bulk of gas takes place by its union with the free ammonia, converting it into a carbonate; whether this is so or not, it is certainly a useful stimulant in this condition. When the accumulation is solid, purgatives, and in acute dyspepsia emetics are useful, ipecacuanha or sulphate of zinc, or both combined. Turpentine stupes are, too, desirable. In connection with this subject is involved that of the relief afforded by retaining the sitting posture, or being propped up in bed, in heart disease, and especially in the acute paroxysms of dyspnœa. There is no question as to the fact. The explanation is not so simply answered; but, after deliberate thought, it appears most probable that the relief is not in the respiratory passages being so brought or kept in a straight line, as has been asserted, as when the patient seeks relief by sitting on one chair, and resting his arms on the back of another (here the fixing of the arms enables the auxiliary muscles of respiration to act more efficiently), or desires to be propped up in bed, or dies sitting in a chair. At other times, when seized with an acute paroxysm, the reclining patient jumps up in bed, gets on his or her hands and knees, retaining this posture for hours, uncomfortable though it is at other times. It lies in the regional relations of the diaphragm. When in the reclining posture, the contents of the abdomen do not fall from the diaphragm by their own gravity, but press equally on the diaphragm as on the other parietes; and this, too, markedly when any load or pressure exists in the stomach or colon. This is at once relieved by elevating the upper portion of the body, or in the attitude on

hands and knees. Whether this is all the explanation or not, there can be no question that it is part of it. All pressure from without upon the heart impedes its action, and on its removal the heart is relieved.

The question of action upon the heart from within, by reduction of the bulk of blood in the venous centres, and especially during distension of the right ventricle, has been discussed and even debated only some few years ago. Most members of the profession will remember the memorable discussion on bleeding betwixt Dr. Markham and Sir Thomas Watson on "The Change of Type in Disease," and will also be more or less familiar with the more recent elaborate paper "On Bloodletting" by B. W. Richardson, F.R.S. The question briefly is the relief of over-distension of the right heart by the withdrawal of blood from the circulation. In cardiac asthma the right ventricle is in over-distension, and at the point of paralysis therefrom, in its inability to drive the blood through the capillaries of the lungs. This condition is unfortunately very frequent in the course of chronic heart disease, and may arise from diaphragmatic pressure, when in the horizontal position, or from spasm of the pulmonary arterioles, &c. Here, again, the question of the relief afforded by the withdrawal of blood generally or locally is not one about the fact: that is unquestionable enough; but whether the immediate danger would justify resort to the measure in the face of the danger looming of oncoming anæmia. A few ounces of blood are not much to a healthy person, but amount to a great deal in a person with advanced heart disease. Consequently, the tendency of practice is to draw blood away from the venous centres by means which do not necessitate its removal from the system. Thus, one method much resorted to is the diminution of the bulk of blood by the administration of purgatives. Another and more speedy plan is the dilating of the innumerable capillaries of the skin by the application of heat, of irritating materials, or both combined. The application of mustard poultices and blisters are measures of no recent adoption; but the advance of knowledge has demonstrated that the effect desired can be more quickly and surely attained by the application of hot

poultices around the trunk, or as far as is practicable. They at once dilate the capillaries of the skin, and thus furnish relief to the gorged venous centres: thus acting in a manner precisely similar to the withdrawal of blood by bleeding. Mustard may or may not be added to aid their efficiency by its irritant action, or by its action as a general stimulant. The use, then, of this method of unloading the venous centres is in practice preferable to that of withdrawing blood from the organism for ever, as is unquestionably the case with general or local depletory measures, and is thus to be resorted to first; but the question of resorting to further measures when these, combined with the administration of internal remedies, fail, is whether the emergency is sufficiently great or not. Several times has the writer stood, lancet in hand, some time, carefully watching the action of the other measures, and, on their success, returned the lancet unused. Though the relief of this condition by venesection must be seen to be appreciated, still the immediate advantages must be weighed against the loss of that blood permanently. No doubt, in practice, local bleeding by leeches is largely resorted to, and so, but for the natural objection to the cutting of the skin, would be wet-cupping; dry-cupping is not so useful here as the covering a larger surface with hot poultices, however useful it may be when applied to a localised surface to divert to the skin the blood of an artery whose internal distribution supplies an internal organ affected, as over the loins in kidney congestion, or in costal pleurisy, &c.

In addition to these measures of acting on the heart from outside and within it, is the question of acting on the heart itself by means of remedies. Can we increase and diminish the completeness and power of the ventricular contractions, is a question which can now be answered in the affirmative. There do exist agents which positively stimulate the heart walls into increased contraction. Thus the condition of the heart walls, as regards distension, is brought within the reach of remedial agents administered internally. The effect of such an agent in the condition described immediately above, is to cause the ventricle to contract more perfectly; and thus it is not only relieved from the danger of paralysis from over-

distension, but aided to recover itself. No sedative to the heart is needed here betwixt its distension and the risk of paralysis from the carbonic acid of the highly venous blood within it; it is only already too powerless, but some agent which will enable it to contract more efficiently. That we are in possession of such agents is certain. The chief one in use is digitalis. The names of Sanders, Orfila, Traube, Dybkowsky and Pellikan, Handfield Jones, Hilton Fagge and Stevenson, Reich, Niemeyer, Winogradoff, are intimately associated with this subject; and though the earliest impression of its action was that it was a sedative, because under it the heart grew quieter, still, from the work of Withering,* in 1785, down to the most recent investigation by the writer,† the idea has gained ground that this quieting of the heart's action arose from its being endowed with more power. Allied to it are other agents, as the scilla maritima, associated with the names of Fagge and Stevenson; the dajasck, or arrow-poison of Borneo (Braidwood), unquestionably belladonna, associated with the names Meuriot, and notably Dr. John Harley; the alkaloid of veratrum, advocated by Trousseau, Aran, Vocher, and Horatio Wood; caffeine, the writer; others of the scrophulariaceæ,‡ and probably scoparium; and possibly some of the trial poisons of the tropics. Physiological experiment demonstrates what clinical observation had long expected, that the effect of digitalis is to produce more perfect action of the ventricles. The condition of complete paralysis by aconite, identical with that of the over-distended heart, was quickly relieved by the administration of digitalis, and not only so, but that previously paralysed heart, in extreme distension, can be brought to a stand-still in the opposite condition of complete contraction, if enough of digitalis be administered. In practice, then, digitalis can be administered with excellent effect in the condition of over-distension of the ventricle, and has been so used. In all con-

* "On Foxglove." Birmingham, 1785. Leipzig, 1786.

† "The Hastings Prize Essay, 1870." "Digitalis: its Mode of Action, and its Use." H. K. Lewis, 1871.

‡ Some experiments by Dr. H. Barnes, of Carlisle, and Dr. J. Wallace, of Dalston, now of Liverpool, on other members of the scrophulariaceæ, may some day see light.

improvement in the circulation is followed by freer circulation through the kidneys and better arterial tension, and, consequently, there is better diuresis of water.

Diuretics have not only always been resorted to in the treatment of dropsy, which was, until recently, regarded as a disease *per se* and not a sequel, but the improved or altered flow of urine has been adopted as a sort of test of the efficacy of the remedial measures resorted to. Long before the experiments of Pnaff, Winogradoff, Traube, Handfield Jones, and Brunton, on arterial tension, popular observation had learnt that an increased flow of urine indicated an improvement in the patient's immediate condition in dropsy; and it is, therefore, in the agents classed as diuretics that we find digitalis, squill, belladonna, &c., which we now know exercise their diuretic action by virtue of the increased arterial tension, the result of more perfect ventricular contraction. The water excretion by the kidneys is a question of hydraulics—a purely mechanical result of the pressure of the blood on the thin-walled glomeruli of the Malpighian bodies. When the arterial blood is driven in but feebly, and the venous is almost stagnant, we find the bulk of urine very low indeed: but its sp. gr. may be very high, for the bulk of urine and the elimination of solid excreta by the renal secreting cells bear no necessary relation to each other. No doubt, too, certain other agents act as diuretics, as juniper, turpentine, potash, nitrate, nitric æther, &c., by their being direct stimulants to the kidneys, and thus inducing an increased flow of blood to these organs. But it is in the first division we find our agents which are the best diuretics in heart failure, and what position scoparium should hold it is difficult to say from want of observation experiments, but it will probably be found among the first named. The other class of diuretics, as colchicum, cantharadine, and buchu, &c., which increase the amount of solids without affecting the bulk of urine, are not to be regarded as diuretics in the treatment of heart failure.* Stimulants are

* The Americans seem better acquainted than we are with the difference betwixt water diuretics and solid diuretics; and this is due to the adoption of

diuretics insomuch as they increase the completeness of the ventricular contractions and improve arterial tension.

Diaphoretics are not yet sufficiently adopted as a means of affording relief in dropsy and the sequelæ of heart failure; but it is not only a very effectual, but, in my experience so far, a perfectly safe way of increasing water elimination, especially when the heart failure is accompanied by renal disease. The sitz-bath is a good form, or any bath which permits the patient to breathe freely: Turkish baths are regarded as not very safe.* Another plan, devised by the late Sir James Simpson, has been so useful that it cannot be omitted, and that is, to fill six or eight lemonade bottles with boiling water, cork them well, and draw over each a woollen stocking wrung out of hot water, then pack them round the patient in bed, who in fifteen minutes or so commences to sweat profusely: they can be continued for an hour, or repeated when necessary. This plan can be resorted to with the poorest patients, and when a patient of any class is confined to bed. In obstinate cases where the skin will not be induced to relax by hot baths, cold packing has been resorted to in order to paralyse the minute vessels of the skin, and thus produce dilatation of them in cases of chronic kidney disease; but I am not acquainted with the details as to whether cases with distinct heart failure were so treated or not.

Dropsy is not only a great cause of suffering to a patient with heart disease, but is regarded by non-professional people as indicating great danger to existence. It is, no doubt, a sign of failing power of serious import; still its occurrence often gives temporary relief. When the distended venous system is relieved by a sudden general œdema, great

the conclusions of Hammond in his essay "On the Action of Certain Vegetable Diuretics," 1859.

* On the other hand, Dr. Bristowe, in Croonian Lectures, 1872 (*Brit. Med. Journ.*, May 11th), says, "I treated my anasarcous patients largely by the Turkish bath; and I was soon surprised to find how well those who were seriously ill bore it, and how much they were, for the most part, benefitted by it. Indeed, I began by degrees to employ it in the treatment of such cases almost indiscriminately, and both for cases of heart disease and renal disease, whether acute or chronic."

and immediate relief is experienced, and that may be utilised to improve matters and to induce re-absorption. But usually dropsy commences insidiously, retreats for a longer or shorter time under proper treatment, but usually it is not long in returning, and is more persistent in each reappearance. If, during the interval, the patient has been under good treatment, and yet, in spite of it, it returns, it is a prognostic sign of the very worst import: no worse sign can exist, and its reappearance calls for the most energetic treatment. Sometimes œdema is very capricious in its seat, and flits about in a curious to-and-fro manner; in one well-remembered case, where the lesion was tricuspid, the oscillations from legs to lungs and back again were very marked If the patient was found with a less agonized countenance, his legs were tremendously swollen, when he was gasping for breath his legs were fallen. Dropsy, as a sequel to heart disease, is amenable, more or less, to the measures cited above, in addition to which is the question of puncture. Nature oftentimes establishes a drain of her own; not always with good effect. Puncture, or incision, is, in my personal experience, in most cases worse than useless, and only adds a wet bed to the patient's sufferings. One case only can I recall where the benefit was decided and unequivocal. Others have had a less unpleasant experience, but it is in dropsy from heart and kidney disease combined that the benefit of puncture or incision is chiefly seen. In simple heart failure it is a hazardous proceeding, and sometimes the carbonic-acid poisoning, the irregularity of pulse, dyspnœa, &c., commences to increase on the establishment of the drain. In one case this occurred when the serum spirted out of the needle-holes in an arterial jet, and the bed was soon in a perfect puddle, and could not be kept otherwise during the six and thirty hours the patient survived. Still, the puncture or incision is among our armamentaria, and does frequently afford more or less relief; but the incisions may become troublesome sores, or even slough.

Another troublesome sequel of heart failure is increased flow of bronchial mucus or serum, a chronic bronchitis or bronchorrhœa. This is not only a result of imperfect nutri-

tion to the respiratory structures and consequent formation of many imperfect epithelium cells or mucus, but is also a consequence of congestion of the bronchial veins, and, as such, gives more or less relief to the distended vessels. It is thus often useful though troublesome, for it disturbs the patient's rest and impedes his respiration. Where this condition is marked, a mixture of digitalis, or belladonna with senega or squill and æther may be prescribed; the two forms given here are useful, one very palatable, and the other very effective.

℞. Tinct. belladonnæ, ♏ xv.
Sp. chloroform, ♏ xxv.
Ac. mur. dil., ♏ x.
Syr. scillæ, ʒl.
Aquæ ad ℥l, ter in die.

℞. Tinct. digital., ♏ x.
Ammon. carb., gr. v.
Inf. senegæ, ℥l, ter in die.

One caution must be observed in the paroxysms of chronic bronchitis in heart failure as regards prognosis. Again and again will such a patient alarm all around him, and even induce his physician to regard dissolution as impending, and in the morning—for it is usually in the evening or early night they come on—fall into a doze, wake up relieved, and go on fairly for some time after. This is, though a fortunate termination, sometimes unfortunate for the physician, in this way :—The alarmed friends sit up all night and next day—the alarm having passed away—are apt to regard the practitioner who, however naturally, led them into error, as wanting in knowledge of his profession. This acute pulmonic congestion, with free flow of serum, must not then be regarded as an accession of acute bronchitis, which would, in all human probability, be fatal, but be described as a period of suffering and peril, from which it is not impossible the patient may emerge. So also may this condition of pulmonic congestion lead to rupture of the vessels and hæmoptysis. This is often very alarming; but this hæmoptysis is not itself injurious, except by the loss of blood; and probably in so severe congestion the loss of blood is necessary, and could not be so cheaply or effectually furnished in any other manner. Blood is blood, and whether it is lost by bleeding, leeches, or hæmoptysis, it is the same. Hæmoptysis is the

most direct form of unloading the over-charged vessels, and is not specially injurious, though, of course, it may go on to a fatal termination, especially when fed by stimulants. But, ordinarily considered, hæmoptysis in heart disease is not itself a disease or dangerous, but it is a symptom of a very serious condition; and it is that condition on which it depends, and not the hæmoptysis itself, which should excite our fears. It is better, too, not to attempt to arrest the flow here by sulphuric acid, opium, and acetate of lead, &c, which certainly disturb the stomach, but to keep the patient perfectly quiet. Should the case be very alarming, it may be desirable to resort to general bleeding.

So, too, when the powers are failing, and the patient utterly worn out, is sleepy to a degree, dozing off for a moment, and then waking up with a start in terror, and gasping for breath—even in this piteous condition to administer soporifics is a moral crime. If the patient sleeps, it is that sleep which knows no wakening, except in another state of being. The late Hyde Salter pointed this out strongly. "Take away," he says, "the necessity for the voluntary effort indispensable to respiration, and the patient would soon be so sound asleep it would be difficult to wake him. Suspend the voluntary efforts by an opiate, and the sleep is the sleep of death." However much we may pity the sufferer, we cannot feel justified in prescribing opium or other narcotic for a condition which is not sleeplessness, but where voluntary effort, incompatible with sleep, is necessary to existence. Nor is chloral hydrate any safer here than the vegetable soporifics.*

The question of stimulants in the treatment of heart disease and its sequelæ is a difficult one. Where they are habitually taken, it may neither be desirable nor easy to withdraw them; but where they, *i.e.*, alcoholic stimulants, evidently increase the rapidity and impair the quality of the ventricular contractions, some attempt must be made to

* Dr. Waters, of Liverpool (*Lancet*, May 4th, 1872), in a lecture on Hydrate of Chloral, says, "In cases of heart disease, where there is albuminuria, I have not seen good results from the exhibition of chloral, and I always hesitate to give it when the kidneys are diseased."

diminish, at least, the amount. Where a dose of alcohol improves the pulse and enables the patient to eat and digest better, its use is indicated. In the most advanced stages, alcohol is simply indispensable, and forms the chief portion of what the patient takes. In the less advanced stages, it is often desirable that the patient take spirit and water before getting into bed; it warms him in the cold bed, and helps him off into refreshing sleep with less evil effect than any other agent. The cold of the bed often has a disturbing effect on these patients. The blood in the skin, chilled by contact with the cold clothes, excites spasm of the pulmonary arterioles by its temperature, and thus taxes the right heart. The bed should be previously warmed.*

External measures are, to some extent, useful—as a belladonna plaster over the region of the heart, for instance, which is a favourite measure with many. It is, to some extent, physically useful; but psychically it often acts charmingly, and so may often be recommended with advantage. As to blisters over the region of the heart to allay irritability, no modern-educated person would think of resorting to them; as good or better effects having been produced in the hands of those who do not resort to them as with those who do, the effect of them may be regarded as adding a troublesome sore to the patient's troubles, and so diverting their attention.

The general treatment, in the more advanced cases, consists of food, nutritious and easily assimilable, in small quantities, and often. Very little or no supper is desirable; and patients soon find out that they must make their choice betwixt suppers and dyspnœa, or the absence of both—the relation between the two is unequivocal. The morning is

* In discussing the effect of alcohol in arresting or relieving intermittent pulse—usually a serious indication of heart inability—Dr. B. W. Richardson says, "If after great fatigue, or excitement, or anxiety, there is restlessness, sleeplessness, and painful knowledge, on the part of the patient, of the hesitation in the circulation, half an ounce or an ounce of brandy will act, generally, in the most effective manner. It will bring rest at once, and often, when a narcotic fails, sleep." Dr. Richardson attributes the effect of alcohol in feeble heart on the brain to its increasing the heart's action, and thus supplying the brain with more arterial blood; and that it acts as a narcotic by virtue of this action; and with this opinion I quite agree.

the time during which the bulk of the food must be taken. The clothing must be warm, for the tendency to lowering of temperature is marked. Exercise must be limited and exertion forbidden. The bowels must be regulated, and no straining at stool must be permitted. Death on the night-chair is not at all uncommon in heart disease. The venous congestion leads to gastric catarrh, with its pathognomonic symptom of sense of fulness constantly, and to constipation, and attention must always be directed to the condition of the intestinal canal. The bladder must be examined occasionally, and, if involuntary dribbling exists, a catheter must be used; for, as Sir Henry Thompson insists, involuntary micturition is the evidence of a distended bladder and not of an empty one. This attention to the bladder is more necessary, as prostatic enlargement is common in elderly men, who are also often the subject of heart disease. But there is another reason, and that is, that, in consequence of the venous congestion of the brain, the patient becomes altered in his cerebral manifestations, and not unfrequently his habits change somewhat. This effect of congestion of the cerebrum and its mental consequences are more frequent than is generally thought. The patient has a tendency to become petulant, whimsical, and childish, and must be treated with great considerateness. The appetite is often capricious, and must be humoured as far as is safe. The whole management of the sufferer from heart disease of an advanced type is especial, and consists of a union of judicious firmness, perfect openness, and infinite patience. The patient must be made as clearly aware of the nature of his case as is possible; and in those cases where the patient positively declines to know anything about himself, the friends should have the case explained to them, for his guidance, as to what he must do and what he must avoid, and why and wherefore. For, if the case is a prolonged one, everything to facilitate a good understanding and perfect mutual confidence must be cultivated; for this mutual confidence will many a time and oft be strained, partly by the oscillations of hope and despair in the patient, but more frequently from the interference of others.

Intercurrent disease of every kind, and especially bronchitis, which taxes the heart dreadfully, is attended with more than ordinary danger, and special attention must be paid to the heart, and measures taken to sustain it, or the patient will surely die. Finally, every case is a subtle problem to be solved; whether it is in that stage that recovery or repair is possible, or in that more advanced condition where treatment can only be regarded as a means of procuring more perfect euthanasia. When, however, the tricuspid becomes obviously affected, whether early on in the case or after a long period, the case passes quickly into the last category. Here no means of acting on the heart itself can do much, and that little only so long as the veins will or can sustain the pressure thus increased. Relief of the venous congestion, by acting on the various emunctories, is all that we can fairly hope for here; and whatever measures be adopted, it is not for long. Whatever hopes of prolonged life had previously existed must be given up, when the tricuspid lesion becomes marked; and from the last division of treatment alone can anything almost be expected.

CHAPTER XI.

AFFECTIONS OF THE PERICARDIUM: ACUTE PERICARDITIS—PATHOLOGY—SYMPTOMS—TREATMENT—PERICARDIAL ADHESION—HYDROPERICARDIUM, &c.

THE pericardium or serous sac of the heart is occasionally congenitally wanting. It consists of two layers, an external and internal one; the latter, closely investing the substance of the heart, extends upwards nearly to the top of the aortic arch when it becomes folded back, and forms the external layer. This serous sac enables the heart to perform its necessary movements without friction, and may contain normally a small quantity of fluid. This serous nature renders it liable to inflammations of both an acute and chronic character, especially in connection with general constitutional conditions. Various chronic conditions result from acute inflammation, from the formation of fibrinous bands betwixt the two pericardial layers up to complete adhesion. In other cases the effusion persists and assumes some chronic form. The pericardium may also be the subject of passive exudation without inflammation at all; this is hydropericardium.

Acute Pericarditis.—This is not an uncommon affection, and has attracted a great amount of attention from the majority of observers, in consequence of interesting peculiarities in its course. When the pericardium first becomes inflamed it presents the appearance of general redness with ecchymosed points, often of a tree-like character. There is swelling in the serous and subserous coats, and the formation of delicate, villous projections and effusion of a serous fluid usually containing shreds and filaments of lymph. This fluid varies in amount and quality. Frequently it is in considerable amount and distinctly fluid; at other times it is more decidedly albuminous, and so dense as almost to possess consistency, and the two layers are adherent. When

separated, the surfaces present a honey-combed appearance, or, indeed, extremely like "tripe," and are not at all unlike two buttered surfaces when applied to each other and then separated. At other times, and when associated with dyscrasial affections, the exudation is bloody and then the pericarditis is termed hæmorrhagic.

Pericarditis has various terminations, and is rarely itself a cause of death. Absorption may take place and extend to a *restitutio in integrum*. While the fluid is absorbed, in other cases the fibrinous shreds form bands which bind the two layers of pericardium together, but, being dragged on by the heart's movements, in time fibrinous bands are formed like pleuritic adhesions. At other times the adhesion is more general, and may extend to a complete adhesion of the two pericardial surfaces and obliteration of the pericardial cavity. The effused fluid in other cases itself undergoes changes, or may even remain simply unabsorbed. The fluid may become purulent and resemble the contents of the pleuritic cavity in emphysema. Under these circumstances, perforation apparently by abscess may occur through the chest walls, and thus may be added pneumopericardium, with the formation of ichorous pus. But more commonly, while the fluid contents of the sac may be absorbed, the more solid ones remain and undergo various changes. The lymph may undergo fatty degeneration, and remain in cheesy or even mortar-like masses, or the more fluid parts of the degenerated mass may become absorbed, and chalky masses remain. These earthy constituents may, along with certain connective tissue corpuscles, become more or less organised, and an ossification, or perhaps more truly, petrifaction may result. This may even be general, and the heart be enclosed in a calcified outer coat instead of its serous coverings.

The pericardium may, like other serous surfaces, become the subject of grey or miliary tubercle, and be studded with it. It may even have deposits of the larger masses of yellow tubercle.

In connection with pericarditis must be mentioned the "milk spots," or "white patches" of the heart, which were at one time thought of some importance other than patho-

logical. They were supposed, from their frequency in soldiers, to result from the "cross straps" which traverse the region of the heart, and were accredited with causing the deaths, or at least aiding in causing them, of those in whom they were found. But more careful investigations have acquitted them of any dangerous or lethal properties, and they are now known to be very innocent growths of white connective tissue immediately beneath the cardiac pericardium, utterly free from any effect upon the tissues below them.

Pericarditis is usually accompanied by more or less extension of the inflammatory process into the muscular structure of the heart. This may extend more or less deeply, but is usually superficial, and is denominated myocarditis acuta parenchymatosa. At other times it extends sufficiently to cause yielding of the heart fibres and the formation of dilatation. This may result from the muscular fibre becoming infiltrated with serum, without necessarily true carditis having extended through the muscular wall.

Very commonly, too, endocarditis is found along with pericarditis, but they cannot be regarded so much as the result of one another, as the results of some exciting force common to each as a provoking cause. The external layer of pericardium may become affected simultaneously with pneumonia or pleuritis, one running into the other by contiguity.

Oppolzer divides pericarditis into four divisions:—

1. Idiopathic, or simple, in which it is unconnected with any constitutional condition. A precisely similar state occurs in the rare cases where it is traumatic in its origin.

2. Consecutive, where it is consequent on some previously existing affection, and has spread by contiguity as when it results from pneumonia, pleuritis, necrosis, or caries of the sternum or ribs, spinal caries, aneurism, &c.; from tuberculous masses or cavities in the lungs, abscesses, &c., or, indeed, any condition which may lead directly to inflammation of the external layer; or from myocarditis which may lead to changes in the internal layer first.

3. Symptomatic. In this division are placed those in-

flammations of the pericardium which accompany rheumatism, Bright's disease; zymotic affections, as typhus, small-pox, scarlatina, cholera; constitutional conditions, as tuberculosis, syphilis, chronic alcoholism, &c.

4. Metastatic, where it is associated with most of the conditions under the preceding division, especially the zymotic conditions, and in addition pyæmia and puerperal fever. The pericarditis of these last conditions is necessarily of a serious character partaking of the general condition.

Pericarditis is frequently associated with scurvy and with purpura, and when so occurring is of the hæmorrhagic character. It has even been found in an epidemic form in Russia, where apparently diseases not usually regarded as epidemic have a tendency to so become, as cerebro-spinal-meningitis, &c. Not uncommonly will be observed epidemic outbreaks; undoubtedly is this the case with hæmorrhagic pericarditis, which has been observed by Russian physicians, on the shores of the Baltic sea, in great frequency in prevalent attacks of scurvy (von Dusch).

But, as commonly and ordinarily seen, pericarditis is found in connection with rheumatic fever. It usually does not manifest itself for some days after the establishment of the fever with its joint affections; at other times it may arise simultaneously with it, or even in some cases precede it. In these latter cases it may form all along the most marked part of the ailment, and it has even been asserted that true rheumatic pericarditis may occur without joint complications at all.

The proportion of cases of pericarditis with acute rheumatism has been variously estimated from so low as 16 per cent. to as high as 75 per cent., the proportion of recoveries being closely allied to this, for where pericarditis is associated with rheumatism it is much less serious than when allied to dyscrasial conditions. Most elaborate tables as to the connection of pericarditis with acute rheumatism have been drawn up by Fuller, and those who wish to enquire further into this subject may advantageously study these tables in his well-known work on "Rheumatism, Rheumatic Gout, and Sciatica."

Pericarditis is rather a complaint of cold weather than of warm weather, and, so, is supposed to be associated with cold, but the connection is not very evident. It is found more in men than in women, but whether the conformation of the female bust has anything to do with this, or not, is not known. It is common in the early years of puberty, and, as associated with rheumatism, is a disease of early life rather than of advanced life. As connected with dyscrasiæ, it is common in more advanced life and in adults.

Pericarditis is a somewhat difficult affection to describe, as regards its symptoms, in consequence of its being so rarely found disassociated from other ailments. Still a more or less approximative description may be given of it. It is usually preceded by rigors, but these are not so marked in consequence of its associations. Its first symptom is usually pain over the region of the heart, not excessive, unless there be also pneumonia or pleuritis, and at times entirely wanting. Still ordinarily there is pain increased by deep pressure. The patient is usually flat on the back, an attitude probably rather due to the general affection of the joints than to the pericarditis. There is a plaintive look of suffering, and of desire for succour, somewhat characteristic. The pulse is accelerated, and at first to some extent bounding, but this characteristic is soon lost, and the pulse soon becomes weak, compressible, occasionally very rapid, at other times not so accelerated, and it is even sometimes delayed; it has a tendency to become irregular, and is ever disproportioned to the apparent activity of the heart. In fact there is often palpitation and considerable cardiac excitement in the earlier stages, but these are merely evidences of cardiac inability. There is decided depression, that is the prevailing characteristic. There are also headache, dizziness, tendency to delirium, especially at nights, and sometimes very active delirium; the countenance is usually injected, and the lips blue. There is increase in temperature, usually not exceeding 104°, but this is a doubtful point also, in consequence of the usual complications. In the latter stages of the fatal cases the temperature may fall below the normal. There is often gastric derangement, in consequence of the unity of

the nerve supply. The urine is small in quantity, usually laden with lithates, and often very free from chlorides, and this, too, is not confined to cases where the lung is implicated; it is often, too, albuminous. There are some peculiar points of interest in pericarditis as regards its symptomatology, to which we will return, and also some curious simulations by it which need notice.

The objective symptoms of pericarditis are the most characteristic points in determining its existence and in separating it from conditions which might be mistaken for it. Indeed, in all cases of acute rheumatism it is imperative every day to examine the heart for physical signs, for all rational symptoms may be absent, or masked. The pain, indeed, may be forgotten by the patient, or masked on account of the excessive pain in the joints, and so also may the palpitation not be noticed.

To inspection there is rarely any sign to be noted, unless the latter stage of effusion be reached, and then it is not easily marked in the adult, though a bulging forward may be discernible enough in the younger persons, whose costal cartilages are yet unossified. Usually in the earlier stages there is no evidence furnished to inspection.

To palpation, however, it is very different. The cardiac excitement in the first stages is to be felt by the hand, and as soon as there is friction betwixt the two dry, inflamed, and roughened, surfaces, this is palpable to the hand. In fact, we may feel the friction murmur, which is manifest to the ear.

This of course may be more or less distinct, according to its extent. When there is effusion this of course is lost, and the heart's action becomes faint or imperceptible. This, however, may not depend entirely on effusion, but may be due to the loss of tone in the heart, and the feebleness of its action.

Percussion gives us no aid in the early stage, except negative evidence of no increase in dulness, or the absence or presence of lung complication. But as the effusion increases percussion becomes of more service; still, like all percussion about the heart, there are many sources of error. At first, when the effusion is limited, it is confined to the upper portion

of the pericardium, the heart occupying the lower portion; and here many sources of error come in, as aneurism, tumours, &c. But as the effusion increases, the dulness assumes a pyriform, or triangular shape, the triangle standing upon one of its sides. This dulness becomes broader when the patient lies down and is less extended laterally when sitting up or standing up.

This dulness is not unlike that of right side enlargement, and there is more difficulty in distinguishing it by reason of this fact, that the right ventricle is often dilated from the effects of the effusion on the muscular fibre. There is one point, however, which is always of value in making the diagnosis, and that is in right side enlargement there is never dulness extending beyond the left apex laterally. Now, in pericardial effusion the dulness does extend to the left of the left apex, and may usually be so detected as deep seated dulness. This will always settle the question, but unfortunately the intervening lung is a sadly disturbing element. The lung may be consolidated, and thus give an impression of extended lateral dulness, or it may be highly resonant, and mask it, or it may be tied down by adhesions, and prevent the changes in amount of dulness ordinarily elicited by testing during a deep inspiration and full expiration. Ordinarily, however, percussion is of great service in determining the amount of effusion, and the errors are corrected by the other means of examination.

Auscultation.—To this means of examination we owe most of our positive information as to the existence and extent of pericarditis. By it we detect and localise the friction murmur which is characteristic of pericarditis. This friction sound (Reibungsgeräusch) is produced by the attrition, the rubbing together of the dry inflamed pericardial surfaces. It is of a decidedly "to-and-fro" character, and is systolic and diastolic. It is usually in proportion to the amount of inflamed surface, and where this is limited may only be heard over a small area. This area of audibility is of importance then in aiding us in knowing to what extent the inflammation has spread. So, when the inflammation has extended over all the surface, the murmur is general, and heard equally dis-

tinctly in all parts. There are, however, difficulties in the way here even, and it is often well nigh impossible to distinguish a pericardial murmur from the murmur of endocarditis so often found along with it, and under similar circumstances. Even the "to-and-fro" character may not be pronounced enough to determine the question, though usually sufficient. When this is the case, the locality of greatest intensity is of great moment, for the endocardial murmur is almost invariably left sided, and if the murmur is heard over the right side of the heart, it is almost certainly pericardial. The murmurs have to be compared otherwise, and Dr. T. King Chambers in his Clinical Lectures advocates strongly the plan of applying the ear to the stethoscope, finding the murmur, and then withdrawing the ear gradually from the stethoscope, retained *in situ*, noting the persistence of the murmur. If heard as long as the heart sounds are audible it is endocardial; if lost while the heart sounds are still audible, it is pericardial. How far this is correct, and has been tested absolutely by the records of the dead-house, I am not in a position to say. It needs investigation.

One thing, however, does not admit of much doubt, and that is that all examinations of the chest, when inflammatory affections of thorax accompany acute rheumatism, should be as brief as is possible and compatible with due care. For the exposure of the chest for any time is always followed by increased pain, distress in the countenance, and general increased discomfort.

It is to be feared that some pericardial affections have taken their immediate origin in the assiduity with which they are sought for, especially in Germany.

Not uncommonly a difficulty arises as to the friction sound being pericardial or pleural. This can be easily determined by making the patient hold his breath, when the pericardial sound can be distinguished. Also when there is accompanying pleurisy or pneumonia, the disturbing element can be always so eliminated.

These various measures, along with the subjective symptoms, will usually enable the medical observer to form a correct diagnosis; but it must be admitted that many peri-

cardial inflammations first reveal themselves to the scalpel. In addition then to the objective symptoms, and the brief account of subjective symptoms given above, some of the more especial points of pericarditis may be discussed. The general depression, especially of the pulse, and its tendency to halt, has been referred to the irritation of the cardiac ganglia, but of this we have no proof; while the evidence is not far to seek that when there is effusion there is pressure on the heart which must interfere with its action, and is sufficient occasionally, when persistent, to induce atrophy of the substance of the heart itself. The extent, too, to which the myocarditis, or even infiltration of the muscular walls, has proceeded, must much affect its power. The difficulty of breathing is also due to vascular congestion of the lungs from the retarded circulation, and perhaps partly to the pain induced thereby from the motion. There is also oppression of the chest and sinking. How far these are all due to the inflammation affecting the large congeries of nerves in the neighbourhood of the pericardium it is difficult to say, but some of the peculiar manifestations of pericarditis are certainly due to its involving nerves in its neighbourhood. For instance, from its action on the phrenic we get the persistent and troublesome hiccup not unfrequently observed. Indeed, it may go so far as to produce complete paralysis of the diaphragm, with falling of the abdomen on inspiration.

It is probable, too, that the inflammation of the portion encircling the aorta may lead to effects upon the numerous nerves there, and produce those gastric symptoms which sometimes entirely take the place of all pericardial symptoms. Acute gastric symptoms have been found to owe their origin in pericarditis, which had been utterly unsuspected previous to the physical examination.

But it is as head symptoms that these irregular manifestations of pericarditis have been most frequently observed. Again and again have all the symptoms of meningitis been simulated by pericarditis. This is of more moment in diagnosis in that rheumatic metastases to the encephalon are not unknown, and are unfortunately almost invariably fatal. Thus it is of great importance to ascertain whether such be really

the case or there is only pericarditis to deal with; for serious as is pericarditis, its fatality is small. There is often acute and wild delirium, and indeed all the symptoms of early meningitis, unless it be ophthalmoscopic ones, and of this we as yet know nothing. The head symptoms often allay all suspicion of anything, but meningitis and the recognition of some sign by which suspicion may be aroused is important. Austin Flint has given this subject careful investigation, and relates two cases at length. Both were typical cases of meningitis, simulated by pericarditis, and in both he observed a peculiarity in the delusion of the delirium. This was the fixed impression of having committed some crime. He says, "a fixed delusion of having committed some crime appears to be a distinguishing feature" (p. 358). This point may turn out to be of practical value in directing attention to the possibility of pericarditis in cases of apparent meningitis. Not only is the prognosis affected by the distinction, but treatment is also directed from a wrong to right neighbourhood.

Terminations.—As might be readily supposed from the position and function of the pericardium, and the various forms of pericarditis, the results of it and its terminations are also varied. Thus the inflammatory action may cease ere the stage of effusion is reached and a complete remission takes place. Or if the fluid contain little or no fibrin, a *restitutio in integrum* is possible. In other cases the fibrin forms bands; or a complete obliteration of the pericardial cavity may, and not rarely does occur, forming what is called "adhesion of the pericardium," a condition frequently not incompatible with some years of life, and which must be considered separately. The fluid may remain, and even become puriform, and open like an abscess; or the more fluid parts may become absorbed, and the solid parts undergoing degeneration become cheesy-like masses, &c.

The effect upon the heart itself varies also. The pressure of the effused fluid may lead to atrophy. The infiltration into the muscles usually leads to dilatation at the time, which may remain in unfavourable cases, but in others is often followed by hypertrophy, which re-endows the patient with a fair share of power and vigour. The adhesions, and more

so if complete, may by incommoding the heart's action, lead to hypertrophy, or in other cases to degeneration and failure. The myocarditis which often accompanies pericarditis may lead to destruction of tissue and heart scars, or contractions. Finally Rokitansky tells us that adhesion may take place along the track of the coronary vessels, and, by constricting them, cut off to a large extent the blood supply of the heart, and lead thus directly to fatty degeneration.

Prognosis.—In forming a prognosis on so difficult a matter as pericarditis great caution and circumspection is imperative. Not only must the amount of inflammation and its immediate complications be considered, but its causational relationships must be carefully weighed. For these affect the prognosis most materially, not only as general conditions themselves, but as also affecting the nature of the effused fluid. The nature of the effusion obviously is of great moment in forming the prognosis, and where it is hæmorrhagic, or purulent, there is a much worse prospect, infinitely worse, indeed, than where there is mere serous fluid with shreds of lymph. Of course the issue of the pericarditis hangs greatly on the nature of the accompanying affection in many cases, and in pyæmia it is of little moment how favourable the pericarditis may be in itself, the condition with which it is allied, or rather on which it depends, is one admitting of little hope. Each case must, from a prognostic point of view, be regarded in relation to itself and its surroundings. But pericarditis accompanying dyscrasial conditions is ever grave and serious, while the pericarditis which accompanies rheumatic fever is rarely fatal. Such changes may, indeed, be inaugurated as are soon incompatible with life, and in this lies the sting of both endocardial and pericardial inflammations. The patient rarely dies in these rheumatic inflammations at the time, but lingers, crippled and wounded, through a more or less brief period of suffering until carried off by the secondary affections.

Treatment.—The treatment of pericarditis is indeed a tangled skein, for while we have still hanging over us the shadow, and often perhaps something more substantial, of the old heroic practice, we are entering a phase of compara-

tive scepticism, or even, perhaps, a new phase of mischievous remedial interference. It is somewhat difficult then to indicate a line of treatment which will steer clear of both old and new dangers. General bleeding for the relief of this condition is now universally abandoned, nor can we feel surprised when we regard the great tendency to depression which is the main characteristic of the complaint. So also, almost universally, is the application of a blister, which was once applied under some impression that it allayed the irritability of the heart. It is possible that the effusion from the cutaneous branches of the internal mammary relieved the pressure on the internal distribution, and so tended to starve the inflammatory process; but the addition of the sore and its being in the way of other applications after are positive disadvantages arising from its use.

Leeches are often applied to the skin over the heart, and in strong persons this mode of relieving the fulness of the cutaneo-pericardial distribution is to be recommended. Dry cupping is also admissible with a similar intent; but the most favourite and universal application is the large hot linseed poultice, with or without a facing of mustard. This also acts on the cutaneous vessels, dilates them, and relieves the deeper distribution. It is effective, can be continued persistently, and does not act prejudicially on the skin, and thus interfere with the adoption of other measures, if they are indicated. The poultice should be large, and removed ere cold, for the occasional removal of a cold poultice by a warm one is most reprehensible. The effect of this application in relieving the pain and in diminishing the evidence of inflammation, the friction-murmur, is often most gratifying; and for those who are in general practice, and can only see their patients on somewhat distant intervals, it will be found a most excellent practice to leave directions with the nurse, in all cases of acute rheumatism, to apply hot poultices as soon as ever the patient complains of any pain in the chest, and to keep them applied till the return of the medical attendant. There is good reason to believe that the early application of hot poultices arrests many a pericardial inflammation at an early stage. Of course as to general treat-

ment, much depends on each man's way of treating rheumatic fever, and this is not a subject affording strong evidence of medical unanimity. But certainly the patient should be clothed in a flannel night dress, or, among the poor, a man's flannel day shirt comes in very handy; and sheets ought to be dispensed with and the blankets kept well about the chest. Nothing is so bad as exposure of the chest, or arrest of the general perspiration. Consequently purgatives are distinctly contra-indicated, not only from the risk of starving the patients, for with some persons it is well nigh impossible to empty either bladder or bowels in the recumbent posture, but also from the disturbance entailed and its effect in increasing the rapidity of the circulation. Diaphoretics are in general use, and so are mild opiates to relieve the pain; the combination in Dover's powder has a wide-spread popularity. In strong persons, a few, from three to five, grains of James's powder, or of pulv. antim. co., may be added to the ten grains of Dover's powder usually exhibited at the hour of retiring to rest. The old rule of Graves is one well worth remembering in the administration of the vegetable narcotics, viz., to give the dose at such a time that the exciting action may have passed away, and the narcotic action come into force at the usual habitual time of sleep coming on. To catch this natural hour of sleep so that habit and the action of the remedy may unite, and not clash, is a golden rule in the administration of soporifics. If the reader has not already formed a positive opinion on the treatment of acute rheumatism, it may not be out of place to state that the evidence brought to bear by Fuller, in his work on Rheumatism, in favour of the alkaline treatment, both as regards its effect on the duration of acute rheumatism and its efficiency in averting cardiac complications, is worthy of great attention. It is, perhaps, the most common treatment, in England at least, and has been adopted by the writer. From one to two scruples of bicarbonate of potash, with ten or fifteen drops of tinct. opii in serpentaria or buchu every four or six hours for an average adult, is a good plan. The amount of opium here is not excessive, in the face of the pain of the inflamed joints, and when given with an alkali it is not so

active as when given alone or with acids, while it is a most effectual diaphoretic. It is well, too, to dilute the medicine by a good drink of fluid after it; this aids in the absorption through the walls of the stomach. The joints may be wrapped up in flannel gently wrung out of a solution of potash and laudanum, and applied in strips wet enough to stick to the skin, but not so wet as to be sloppy.*

These measures with a milk diet, or mixed with beef tea, will ordinarily be found efficacious and satisfactory. As soon as the activity of the attack is over, the potash may be somewhat reduced in quantity, and some potassio-tartrate of iron added. The usual plan of treatment of convalescents by tonics, good diet, and cod-liver oil where necessary, may be followed here, and needs no comment, unless it be to always push the treatment, when any of the joints of the hands swell and are painful; to lose time then is unfortunate. If the threatened pericardial affection be altogether averted, or pretty effectually restrained by the measures adopted, the case usually progresses favourably to complete recovery.

Before, however, proceeding to describe the treatment of remaining effusion, it is almost necessary to say a word on the German plan of the application of ice to the precordia or pericarditis. We could almost tell previous to applying it, that this contraction of the cutaneous branches, so induced, would tend to drive the blood in greater force into the internal distribution. This may relieve the pain and place, as Hilton calls it, the inflamed pericardial surfaces in physiological rest, by separation, but it may be gravely questioned if this is a result to be wished for. Certainly the numerous cases of death produced from the consequences of cardiac complications of acute rheumatism, in young persons, to be seen in the Pathological Institute of the Vienna Krankenhaus are not very convincing arguments in favour of the plans of treatment adopted. Niemeyer says "Calomel and blue ointment, in spite of the praise of English physicians, are not only useless, but hurtful." While admitting to the full the probable truth of this, I must also be permitted to most gravely call in question the plan he recommends of the appli-

* The blister treatment of Herbert Davies is also largely adopted now.

cation of cold. The results are not by any means gratifying, and several who watched this plan in the Bellevue Hospital, in New York, inform me that its results were decidedly unsatisfactory.

The practice originated in the good results sometimes observed from the application of cold to the chest in hæmoptysis. But the anatomical relations of pericarditis and of hæmoptysis to an ice bladder on the chest are too different for similar results.

Where there is great depression and the pulse is failing, with other evidences of depressed circulation, both Niemeyer and Oppolzer recommend strongly the administration of digitalis; and certainly its use under these circumstances is clearly indicated, along with stimulants and tonics.

Where effusion remains in the pericardial cavity, the usual measures are diuretics, purgatives, and the internal administration of iodine.

The measures are commonly successful, and are usually combined with the application of blisters over the cardiac region. The use of blisters at this time is attended with more favourable results than when applied during the acute inflammatory stage. It has been seriously disputed whether the absorbent action is during the healing of the blister, or continues during the time of the sore, which has therefore been artificially prevented from healing. The more usual plan is to apply a blister, and repeat it when the surface has healed. The old cantharides blister was used, but now the stronger preparations of iodine are preferred, for various reasons.

These measures, however, may fail, and the pressure on the heart may demand that some operative procedure be resorted to.

For evacuating the fluid directly, the operation of paracentesis pericardii has been resorted to, and that, too, successfully. Dr. Clifford Allbutt has advocated this plan, and set the example of resorting to it in practice. From the tendency to the formation of ichorous pus on the admission of air into the pericardium (pneumopericardium), the extraction of the fluid by suction, as by the pneumatic aspirator, so as

not to admit of the entrance of air, is desirable. It is not, however, a proceeding to be lightly undertaken, and on one occasion at least, and in competent hands too, it is recorded that a dilated heart was itself tapped. This shows how difficult it is to ascertain exactly the position of parts within the distended pericardium, and how carefully must the examinations be conducted previous to inserting a trocar into the thorax in the cardiac region.

Still, the practice is one which is likely to be more largely adopted, as, when serous membranes have once been changed by inflammation, they do not readily put on again acute inflammation, which used to so alarm our predecessors in attempting any measures which might excite inflammation of a serous membrane. For the relief of purulent pericarditis older surgeons have even trepanned the sternum, and that, too, successfully.

For the treatment of pericarditis occurring in dyscrasial conditions, it is difficult to arrange anything like a rule. It must here so obviously depend on the condition along with which it occurs, that any special treatment is well nigh impossible, and any local treatment even must be dependent on the treatment of the general condition. Stimulants must be largely resorted to after effusion is established, but during the dry stage it is doubtful, if any good could counterbalance the increased rapidity of the pulse and the increased friction thus necessarily entailed.

Pericardial Adhesion.—One of the common consequences of pericarditis is adhesion of the pericardium, a condition however not incompatible with a more or less prolonged existence. The amount and extent of adhesion are varied, and exert a decided influence over the progress and prognosis of the case.

The adhesion may be partial, and consist of one or more adhesions drawn, by the heart's action, into bands, and resembling pleuritic adhesions.

There may be adhesions dividing the pericardial cavity into loculaments containing fluid or other contents.

The adhesion may be complete, and the pericardial cavity completely obliterated.

This adhesion may be complete, and also contain the

chalky *débris* of the fibrin of the pericarditis, or a sort of union may have taken place with some connective tissue corpuscles and a calcified ring, apt of old to be termed ossification, may be formed.

This adhesion may extend to the costal pleura; and under these circumstances only are there any objective symptoms of its existence.

These different forms of adhesion exercise different effects upon the muscular structure of the heart itself. The chief changes are hypertrophy in favourable cases, and in good nutrition, by which the incommoded heart is able to fulfil its function for some years fairly efficiently. In other cases the tendency is to dilatation where there is not perfect adhesion, which would tend to prevent it.

In other cases, however, the muscular fibre is discoloured, pale, and the subject of fatty degeneration. There are rarely wanting traces of myocarditis with its scar-like depressions. There is also a tendency for the inflamed pericardium to adhere along the track of the coronary vessels, and when this occurs there is rapid and extensive degeneration of the muscular structure from the diminished blood supply.

Symptoms.—The subjective symptoms of adherent pericardium depend solely on the condition of the heart with which it is associated. Where there is fair hypertrophy, the patient may exist years without any evidence of its presence. Thus Stokes had a case which existed seven years without any evidence of its existence. But the cases where it is accompanied by evident degeneration of the heart wall are also accompanied by evidences of heart failure. These are, difficulty of breathing easily aggravated, evidences of general venous congestion with injected countenance, abdominal plethora, and dropsy in time. There are, too, attacks of angina pectoris. Thus, though there are no positive subjective symptoms, when these symptoms follow after general pericarditis and regularly increase in severity, there is presumptive evidence that pericardial adhesion exists.

Objective Symptoms.—When the adhesion is confined to the pericardium solely, there are no objective symptoms to be relied upon by which it may be recognised. Skoda says,

"no symptoms are discoverable, through percussion and auscultation, which can be ascribed to adhesion of the heart and pericardia." Nor to their auxiliaries, in examination, is much divulged. It has been stated that there are loss of apex-beat, and where still observable, no changing of its seat on change of posture or movement of body; but these are to be entirely relied upon for a diagnosis.

When, however, the pericardium is adherent to the costal pleura, there are signs of this which are of positive value. The adhesion to the pleura furnishes to inspection a retraction of the intercostal spaces on each systole. The heart becomes shortened on each systole, and so the intercostal space is dragged inwards on each contraction, and expands again when the heart becomes longer on diastole. To palpation too is felt this retraction along with what Hope called a "jogging motion" of the heart. This sign is regarded as of some value. Bouillaud says, "On sent à la main que la jeu du cœur est embarrassee."

To percussion, too, there are some signs, the most important one being the non-alteration in size of the area of percussion dulness in expiration and inspiration in comparison to the normal.

Prognosis.— The prognosis of pericardial adhesion is hopeless as to cure, and bad as to duration of life. When it is positively diagnosed, a doubtful existence is all that can be hoped for, and the consequences of it upon the heart walls will show themselves sooner or later. But when it is accompanied by presumable hypertrophy, as evidenced by a fair pulse, good general health, and fair physical power, it would be rash to hazard any opinion as to the probable duration of life. When once evidences of the degeneration of that hypertrophy show themselves, no great prolongation of existence is probable, or can be hoped for.

Treatment.—The treatment of this condition does not consist in any attempt to affect the adhesion by any agents, but merely in attending to the symptoms which show themselves, and in the appropriate measures for preserving a fair condition of the muscular walls. Thus exercise and exertion must be limited so as not to tax the embarrassed heart, while

nourishing diet, preparations of iron, &c., are absolutely necessary. For evidences of heart failure the same measures must be resorted to as when they occur without pericardial adhesion.

Hydropericardium.—Hydropericardium or the effusion of serum into the pericardial sac is a non-inflammatory complaint, and is not to be confounded with the effusion following acute pericarditis. It is not associated with any inflammatory process of a chronic character, but is a serous effusion depending on cardiac or renal disease. It is a mere effusion without lymph, and when absorbed leaves no trace behind it. It has at various times been chemically examined, and a recent analysis by Wachsmuth gives the following result:—

In each 100 part of effusion—

Water from	95·37 to 97·34
Fixed material from	2·66 to 4·63
Albumen from	1·43 to 3·01
Other material from	1·23 to 1·64

It is often extensive in quantity, and is a source of distress to the patient, though not commonly a source of danger. In consequence of the limited space, the effusion does not usually amount to more than five or six ounces: though it may amount to three times as much; small quantities below an ounce can scarcely be regarded as pathological.

The effect of this fluid upon the heart is of course to embarrass its action, while the heart fibre itself is pale and easily torn. This does not arise from any degeneration so much as infiltration with serum and impaired consistence. It may by pressure lead to diminution of bulk in the heart itself, but that is rather a result of the effusion of pericarditis. The fat of the heart, however, disappears in proportion to the duration of the effusion (Rokitansky).

The conditions under which it is found are:—

1. Scarlatina. It is a most common concomitant of the œdema which frequently follows scarlet fever. This depends on the blocking up of the renal uriniferous tubules with epithelial casts in the desquamative stage of the tubular

nephritis which usually accompanies scarlatina. It comes on insidiously, and is ordinarily found along with pleural effusion. It is entirely a passive exudation, and is soon reabsorbed on the renal flow being again established.

2. Bright's Disease. It is also by no means rare in the latter stages of chronic kidney disease where there is diffused œdema. No attempt has been made to show that under these circumstances the fluid contains urine salts, though it is not improbable. When occurring in Bright's disease is more serious and more obstinate than in the preceding division, and altogether indicates a more serious condition.

3. It is also found as a result of chronic heart disease, and then, like most serous effusions, is only found in the latter stages of the case. Its presence here by further embarrassing the already failing heart is a very serious matter, and adds much to the patient's sufferings. When occurring under these circumstances, it is the result of the general venous congestion, exactly like the other serous effusions, with which indeed it is identical. It may seem almost unnecessary to lay stress on this, but it has been stated by several writers (Niemeyer, Oppolzer) that it is the result of engorgement of the right heart, and effusion from the coronary veins and the cardiac pericardium. This is highly improbable, for, in the first place, such regurgitation is specially provided against by the manner in which the coronary veins open obliquely through the wall of the right auricle, thus acting in a valve-like manner; for the greater the distension the more the edges of the veins are closed together and reflux prevented. Such regurgitation of venous blood into the heart would produce more serious consequences even than effusion through the cardiac pericardium. The effusion takes place from the veins of the external pericardium which partake of the general venous engorgement in this condition of enfeebled circulation.

4. It may occur with a general dropsical condition, whether in acute dropsy or in the more chronic conditions of dropsical habit, not yet understood pathologically.

5. Niemeyer is inclined to regard one form of hydroperi-

cardium as a species of compensatory effusion. He says, "We have already seen how a decrease in the size of the heart, by reducing the pressure upon the pericardium from within, results in an increase in quantity of the liquid in the sac. The same thing takes place when the lungs become adherent to the pericardium and are reduced in volume, either from atrophy, failure to regain their normal size after absorption of a pleuritic effusion, or contraction from chronic pneumonia.

"This form of hydropericardium is analogous to the increase in the amount of cerebro-spinal fluid which takes place in atrophy of the brain, and, as the latter is called hydrocephalus *ex vacuo*, so hydropericardium *ex vacuo* would be a suitable name for the former."

6. It has been found along with miliary tubercle of the pericardium, in acute tuberculosis. Here it has no interest other than pathological.

Symptoms.—The general or subjective symptoms of hydropericardium are those of impeded circulation and respiration. These may, however, not be marked. As for the more general symptoms these depend chiefly on the circumstances with which it is associated, as in scarlatina, chronic heart disease, &c.

The symptoms due to the effusion are often nil, but when it is excessive, as it is in the latter stages of heart disease, it adds to the dyspnœa and forbids the horizontal posture, compelling the patient to sit in a chair, or be propped up in bed. No doubt the weight of the pericardial fluid in this position falls away from the great veins, and thus gives relief.

The objective symptoms are diminished apex beat, and somewhat of bulging of the chest wall in younger subjects, and loss of intercostal depressions. The impulse of the heart is feeble, and Walshe thinks its apex is somewhat tilted upwards. On percussion there is increase of cardiac dulness, increased laterally on lying down, and resembling to some extent a triangle resting on one of its sides. But percussion here, as usual, is apt to be interfered with by conditions of the lung. On auscultation the heart sounds are heard clear

and distinct, but not loud; there is no friction sound at any time.

Prognosis.—In scarlatina this is ordinarily good; in chronic kidney disease it is certainly serious; but in chronic heart disease it is bad indeed, and is usually a precursor, though it can scarcely be called the cause, of death. In the other conditions the prognosis in each case will depend much on the peculiar circumstances of each case. In chronic lung disease the hydropericardium *ex vacuo* is not likely, to say the least of it, to pass away; in the dropsical habit it would entirely depend on the general condition.

Treatment.—This is by acting on the cause where practicable. In scarlatinal dropsy the idea of Dickenson of washing out the renal tube casts is well founded, and the achievement of this by copious draughts of fluids is aided by the use of digitalis to increase the arterial tension, and increase the flow in the glomeruli of the Malpighian bodies. Blisters may be used over the cardiac region if necessary. In chronic kidney disease purgatives may be resorted to when the renal secretion is defective, and more so when of low specific gravity, and these, too, brisk ones.

Potash, colchicum, buchu, and juniper may also be used to act on the kidney, when all active congestion is past. Hot-air baths, and other sudorifics are desirable. In chronic heart disease digitalis may be increased in quantity, or, if not previously used, resorted to, but it is merely as a palliative in the great majority of instances, for any hope from treatment will usually have passed away ere this stage is reached. In other cases the treatment must be guided entirely by the peculiarities of each case, and where a young practitioner does not see his way, the most direct and effective plan is to call in the aid of another head.

Hæmopericardium.—This may occur from some injury to the pericardium, and when so occurring, is usually absorbed, or it may occur in rupture of the heart walls, where it is useless to attempt remedies. It is recorded, however, that in one case of rupture into the pericardium the patient lived three days, and certainly clots have been found in the tears, as if some attempt at repair was attempted.

Pneumopericardium and Pyopericardium.—These two conditions are usually found combined, though one may precede the other. The combined condition may arise either from suppuration within the pericardium making its way outwards, or from an abscess opening into the pericardium, and gas being evolved from chemical changes in the pus. It may be in the lung and the air thus admitted with the matter. The evidence of this accident occurring, and of air in the pericardium, is the clear note, indeed tympanitic on percussion. The tinkling of succussion is also heard. On these two witnesses, in addition to what has gone on before, and the collapse accompanying it, the diagnosis is based.

For the relief of this condition, Bamberger and Friedreich recommend the use of an exploring trocar, and the subsequent injection of chlorine water or iodine. Nor is it easy to see objections to this, as the already inflamed serous membrane will not again put on acute inflammatory action, so justly dreaded in a serous membrane not previously altered by inflammation. Niemeyer relates a case of pyopericardium, arising from cancer of the œsophagus, which occurred in his clinic, and regards the treatment as consisting chiefly of the administration of stimulants, which would alone be of service in such a case.

Growths in the Pericardium.—In addition to the development to tubercle, the pericardium is liable to become the seat of cancer, both carcinomatous and medullary. Hydatid cysts of the pericardium are not unknown.

CHAPTER XII.

Nervous Disorders of the Heart—Angina Pectoris—Nervous Palpitation—Irritable Heart—Sub-Paralysis—Hyperæsthesia—Graves' Disease—Chorea.

Angina Pectoris.—Angina pectoris, or "breast pang," is also denominated neuralgia cardiaca, hyperæsthesia plexus cardiaci, and is usually classed among the neurosal affections of the heart; Handfield Jones and Romberg include it among the affections of the nervous system. There is no agreement among writers as to its pathology as a nervous affection. Romberg calls it hyperæsthesia of the cardiac plexus; Bouillaud, a neuralgia of the phrenic nerve; and Heberden, cramp of the heart.* On the other hand it is admitted that it is most commonly found in fatty degeneration of the heart, and in ossification of the coronary arteries, the combined condition being most frequently found along with it. Fuller states that it is never found without structural changes to be detected by the microscope. The best description of it is probably a neurosal affection, occurring most commonly in structural disease of the heart. It may somewhat elucidate this difficult subject to see what angina pectoris is not, or probably not; its pathology is not a subject for bold expressions or dogmatism.

An attack presents the following features, sudden oncoming, with intense pain in the breast, extending down the left arm, the pulse is increased in frequency most remarkably, the heart's action is feeble, and scarcely to be detected, the pulse is small, rapid, and almost imperceptible. In addition the features are white, with a look of alarm on them, and the sweat rolls down the brow in the large bead-like drops, usually associated with intense agony, mental or bodily. The breathing is shallow, hurried, but not laboured, the

* Anstie says it is "a mainly unilateral morbid condition of the lower cervical and upper dorsal portion of the spinal cord."—"Neuralgia," 1871.

patient appearing rather to not dare to breathe than to have difficulty in doing so. The attack passes off with eructation of wind, vomiting, or evacuation of the bowels, and commonly a large quantity of pale-coloured urine, almost like water. During the attack the patient preserves the same position as occupied when seized, and dares not be moved, the suggestion to move him being pain to him and causing terror. Here now we have some symptoms which cannot be explained by the theories yet enunciated. The signs or objective symptoms do not correspond with stimulation of the vagus; here the heart is slowed remarkably, and section of the vagus gives irregular and tumultuous action. It is not hyperæsthesia of the cardiac plexus, for the heart's action is feeble and undiscernible, and not excited; it is not cramp, for that is excessive ventricular contraction, if ever it occurs, and in this condition, in digitalis poisoning, the heart has a thud, is slow, and irregular, and there is complete, or almost complete suppression of urine. As to its being neuralgia of the phrenic, it is difficult to see the connection, especially with the structural disease of the heart in which it is most frequent. The pain, too, is rather a sickening, depressing pain; which is the character of pain, when it is received as a sensation through the sympathetic.

There is one point on which almost all are agreed, and that is the great lowering of the heart's action, while one point of interest, viz., the condition of the heart after death, corroborates this. Von Dusch says that without doubt the abolition of the heart's action is the cause of the death resulting from it. This effectually disposes of the theory of cramp, and of hyperæsthesia of the cardiac plexus. It is evidently not due to an effect through the vagus, that would give the opposite symptoms of retardation and long halts. Experiments on the accelerator and depressor nerves of the heart in animals will not account for the symptoms, though there is great acceleration in irritation of the accelerator nerve. It is nervous, and yet connected with structural disease of the heart, ossified coronary arteries, and aortic atheroma. It seems most probable that it is a disturbance of the vasomotor nerves within the heart. As a purely nervous affec-

tion it is often connected with uterine, renal, and intestinal affections. With these organs it is connected by the sympathetic: the character of its pain shows its association with the sympathetic. It is allied, in nature, to the arteriole spasm of hysteria and Bright's disease, the two affections with which it is most frequently found, in the one as a purely neurosal affection, the other as a neurosal affection intimately associated with a structural disease.

In chronic Bright's disease, as we shall see at length in a following chapter, there is a thickening of the muscular walls of the arterioles, a true hypertrophy of their muscular tunic, as shown by Prof. Geo. Johnson, of King's College.

Spasm of these thickened arterioles is associated with the cerebral anæmia so commonly found, with the dry harsh skin, muscular pain, or when continued, wasting, and with the white "dead" hands and feet, all common in chronic Bright's disease.

There are stronger grounds for supposing angina pectoris to depend on spasm of the arterioles of the heart, than are to be found in favour of any other pathology. When too complete, death results; when incomplete, an approach to it, with a distinct sensation of its nearness. This condition is equally reconcilable with its being reflex spasm from irritation, as when uterine at the menopause, a common period for it in women, and passing away; or hysterical, as in younger females; and with its frequent accompaniment of chronic structural disease. But it may possibly arise from defective blood supply, not being due to thickened arterioles, for in the condition of ossification of the coronary arteries a disturbing neurosal cause would, by producing spasm of the arterioles, more effectually cut off the blood supply, than when the arteries were sound and aiding in the arterial circulation. Limbs perish of senile gangrene, on ossification of their nutrient arteries, from imperfect blood supply. So in angina pectoris, causes which would only induce an arteriole spasm in the healthy coronary circulation so imperfect as not to constitute angina would in disease of the coronary arteries produce more serious effects.

Arteriole spasm would more completely arrest the blood

supply in this condition than when the circulation and its vessels were healthy. So we may safely assert that in ossification of the coronary arteries, angina pectoris, if not more easily induced, may be induced by alterations of the calibre of the arterioles, which would not be so apparent in healthy persons. That is granting that angina pectoris is the result of spasm of the arterioles; but this cannot be regarded as certain—it is merely highly probable. Actual experiment of partial ligation of the coronaries and a record of the result and symptoms would be most interesting, and would either corroborate or disprove this view. That angina pectoris is due to defective blood supply from arteriole spasm seems highly probable. In one truly neurosal case, which I had the opportunity of frequently observing during the attack, the later attacks terminated in an epileptiform condition, a condition shown by Schrœder van der Kolk to be most probably connected with variations in the calibre of the arterioles.

Age.—It is in the more advanced periods of life that angina pectoris is most commonly found. Forbes found 72 out of 84 cases over 50 years of age. Copland 70 cases out of 100. It is thus associated with that time of life when structural disease in the blood vessels is most common.

Sex.—It is most frequent in men, who are also more commonly affected by fatty heart and atheroma. Forbes had only 8 females in 88 cases. Heberden 3 females in 100 cases. Lartigue 7 in 67 cases, and Lussana had 97 cases all in men.

The pathological changes with which angina pectoris is most commonly found, are thus arranged by Forbes, who in 39 cases found 24 with atheromatous degeneration of the aorta; 16 with ossification of the coronary arteries; 16 with calcification or other disease of the valves; 12 with abnormal softness of the heart.

These figures are taken from von Dusch's work.

All these figures go to prove that angina pectoris is associated with that condition of thickening of the muscular walls of the arterioles, with atheroma of the arteries, disease of the valves, and fatty degeneration of the heart, all of which

are associated with chronic kidney disease, so common in elderly men.

Diagnosis.—We have seen, in a previous chapter, in what essentials angina pectoris differs from false angina, or cardiac asthma, and spasmodic asthma. As seen in the aged and in structural disease, it is a characteristic affection not to be easily mistaken.

In younger persons, especially females, it is scarcely to be distinguished from hysterical asthma, where there is shallow breathing, hurried, small pulse, cold white skin, with rapid and almost imperceptible pulse, and finally discharge of a large quantity of watery pale urine. Oppolzer adds the asthma of hypochondriasis to the asthma of hysteria as a condition from which angina pectoris is not easily distinguished. The intense pain is a point of distinction, but it would be difficult to prevent a hysterical or hypochondriacal person adding that to their sufferings on the slightest hint of such a thing: the question should be put negatively in all cases of suspicion. W. T. Gairdner has described a condition of "angina sine dolore," where all the symptoms of angina are present, but without the peculiar breast pain. Pain down the left arm may occur with angina (and passes down the arm to a finger often, or alone), and has been deemed a sort of very imperfect angina, but its diagnostic value in heart disease is not decided upon. In all cases of angina a most careful examination of the patient must be made, as soon as conveniently may be, after the attack, and the condition of the circulatory system keenly scrutinised, to determine whether the angina be simply neurosal, or a neurosis associated with a very serious form of organic disease. This is most important in forming a prognosis.

Prognosis and Terminations.—When simply neurosal, and, during the intervals, the heart's action is normal, and its sounds and impulse are good, angina is not a complaint with a bad prognosis, serious as the attacks appear, but often wears off, especially when occurring during the change of life. But when associated with organic disease of the heart and blood vessels, its import is very serious. The first attack has been known to be fatal. In other cases the second. In some cases the attacks recur again and again, the intervals

becoming shorter, and the condition during the interval more and more impaired, until at last one proves fatal. When the muscular structure is the subject of molecular necrosis, its functional power is much impaired, and the deprival of its blood is followed by most grave abolition of function, if not total cessation of action.

But angina pectoris is not pathognomic of fatty degeneration of the heart, as once thought, though it is very commonly associated with organic disease; more commonly in all probability than the figures of Forbes would seem to indicate, if the muscular structure of the heart were in all fatal cases subjected to accurate microscopical examination.

Treatment.—The treatment of angina pectoris consists of two divisions: treatment during the attack, and treatment during the interval.

When associated with fatty degeneration of the heart, the rules laid down for that affection must be adhered to. When simply neurosal, the treatment consists of removal of all causational conditions as uterine congestion, or ulceration of the os uteri, by their appropriate measures; ovarian irritation or congestion must be relieved; and dyspeptic and other states attended to. Fresh air, plain food, exercise, cold bathing, with proper moral and social management must not be neglected. Iron, zinc, quinine, or arsenic may be given combined or alone. Arsenic has long held a high position as an agent of value in the treatment of simple neurosal angina pectoris. Its treatment is that of neuralgia, with which it is allied. Anstie has shown (on Neuralgia) that it is one of the manifestations of nerve disorder in families, who are liable to these manifestations in one form, or other form, and that it is allied to neuralgiæ regionally near it.

During the attack stimulants, musk, camphor, &c., have been given. Narcotics and antispasmodics, as large doses of opium or morphine, or even subcutaneous injections are not very desirable agents to administer in a condition, whose pathology is enveloped in doubt. Chloroform or other inhalations are not very safe agents, as their action on the heart is not known. Mustard poultices are safe, if they are of any service, while foot-baths, with mustard, are undesirable as

tending to disturb the patient, which is never desirable during the attack: indeed, sometimes the patient may assume an odd attitude or position and retain it. Digitalis during the attack has seemed to the writer to afford relief. Certainly stimulants seemed to have more effect on the attack after digitalis was added.

Brunton gave nitrite of amyl with good results: as might be expected from its undoubted effect in dilating the smaller blood-vessels.

According to Flint (p. 305), Duchenne and Aran have found the electrisation of the skin in the precordial region remarkably effective both in arresting the paroxysms and postponing their occurrence.

Agents which induce ventricular contraction, or relax arteriole spasm are indicated, and diffusible stimulants of all kinds, from sal volatile to hot spirit and water, are indicated and may be safely administered.

Nervous Palpitation.—Palpitation is usually classed among the nervous affections of the heart, though it is also connected with structural disease, and especially dilatation. Palpitation has been denominated an over-action of the heart, a condition which is not only not proven, but can scarcely exist; for it would simply mean obliteration of the ventricular chamber and approximation of the walls. Then it was denominated a derangement of nerve-balance betwixt the branches of the vagus and the cardiac ganglia, a portion of the sympathetic. This it may somewhat more possibly be, though a condition of palpitation has never been attained in any of the experiments on the ganglia and the vagus. A condition similar, if not identical with it, has been experimentally attained by Cyon, Ludwig, and von Bezold, by irritation of the nerve centres, even when every direct nerve communication with the heart has been severed. Contraction of the arterioles elicited quicker and more excited action of the heart, as a simple result of the opposition offered to the blood flow. And we know from Prof. Rutherford that this is effected through the vaso-motor excitory branch of the sympathetic, which contracts blood-vessels.*

* See 1st Chapter,

Nervous palpitation is in all probability due to arteriole spasm, offering obstruction to the flow of blood, cardiac distension from that obstruction, and palpitation its outward sign, or objective symptom. A condition closely allied to palpitation is found after violent exertion, before the heart has time to settle down on the removal of the call upon it. Violent palpitation is induced in a weak heart by any exertion, and if it were over-action its presence would endow the patient with the very power he lacks, viz., better ventricular contraction; but he is not so endowed, indeed, he is less capable during the palpitation. Violent palpitation was noticed by Hope when the right ventricle was almost paralysed from over-distension, in cardiac dyspnœa. Palpitation in a person quite healthy, but with a weak heart, is induced by any sudden exertion, as running up stairs or after an omnibus. It is obvious that palpitation is not due to nerve disturbance when it is found in conditions so removed from all disturbance of nerve balance. It is equally obvious that it must belong to some condition common to all, and I am firmly pursuaded that condition is cardiac distension, however produced, whether by inefficiency in the muscular walls, or by nervous action, which we now know can act on the heart through the vaso motor nerves of the small systemic arterioles.

We will now see how far this view is borne out by the manifestations of a hysteric attack, where palpitation, truly nervous, is very commonly induced. The patient is pale, with cold hands and feet; indeed, the skin generally is pale and cold, the radial pulse is small and cordy, not unlike the pulse of abdominal inflammation, the carotids are full and bounding, and the heart feels to the hand as if it would forcibly shatter off the anterior wall of the thorax. Along with this there is great secretion of urine, and after the attack a great, often enormous, quantity of pale coloured watery urine is passed. This may be fairly taken to represent the effect of the increased arterial tension than of any nervous action on the kidney. Now we have here evidence in favour of arteriole spasm, the cold extremities, small radial pulse, showing that the muscular coat of even arteries as

large as the radial is affected, while the carotids are full and bounding. The heart is struggling against a general opposition offered to it, and is in the same state as when it palpitates from any other opposition offered to it; and from increased arterial tension, betwixt the struggling heart and contracted arterioles, there is an increased flow of urine. There is some positive evidence here in favour of arteriole spasm, while the other theories have no evidence whatever that can be adduced to corroborate them, the violent action of the heart being no proof of anything but of demand on it, and is induced equally in other circumstances where neither over-action nor disturbance of nerve-balance occur. A symptom common to different morbid states is, in all human probability, due to a condition common to all of them. Palpitation appears due to attempt upon the part of the heart to contract and fulfil its function in the face of opposition. Incomplete emptying leads to over-distension, and palpitation is its objective symptom.

So when a person with a weak, and so-called excitable, heart makes any sudden effort, as running up stairs, an attack of palpitation is induced. This is not a disturbance of nerve balance induced by the act of running up stairs, but the muscular effort makes a demand on the heart, the action of the muscles being to obstruct the blood stream, while they tend to force on the venous blood towards the heart; thus temporary distension of the heart is induced, and a temporary palpitation ensues.

Exciting Causes.—The anatomical causes of palpitation we have just examined, but the inducing or exciting causes of true nervous palpitation are various. First comes the temperament. The subjects of palpitation are usually of the nervous type, or nervous diathesis of Laycock, persons in whom the nervous element preponderates, and who are emotional and susceptible. Thus the nervous constitution of the female sex renders females more liable to it than males. Further, temporary causes affecting the emotional nature increase this susceptibility, as sudden surprise, excitement, anxiety, shock, &c. Certain periods, as the commencement of the menstrual flow, and a short time before it, render

females periodically liable to palpitation. So too at puberty and the menopause, and also after indulgence in stimulants and too much tea, in males tobacco, it is found: here there is exhaustion of the sympathetic, and cardiac inability, so that distension is more easily induced. The effect of ovarian excitement, of too frequent indulgence in coitus, or in substitutes for it, is marked; indeed, palpitation is commonly associated with hysteria, whose pathology—if it really has one, and it is probable—is shown by Dr. Matthews Duncan to be due to ovarian congestion or chronic ovaritis.

Palpitation is connected with disturbances of the sympathetic by disease of any viscus, especially pelvic, and by exhaustion of the sympathetic, however induced.

Palpitation may arise in a weak heart from sudden effort, from any call upon the heart, and of course true nervous palpitation will more readily be produced in those persons than others, so that its production in them by effort at one time, and by mental emotion at another, is quite reconcilable, and is easily comprehended. It is common in the subjects of Bright's disease, for reasons we shall see after, and the changes in the arteriole walls induced by this disease would also render them liable to true nervous palpitation.

Age.—Youth is more subject to palpitation than adult life. It rarely occurs before puberty, except from sudden start, or shock. It occurs in adults in middle life, in women chiefly, and in some nervous men; and in advanced life, when heart disease and Bright's disease are more frequent, it is again found more commonly.

Sex.—Women are more decidedly liable to palpitation than men. This is due to their more susceptible nervous system, their emotional and impressible nature. That arteriole neurosal changes of calibre are more readily induced in women than men is evidenced by the blush, that strange momentary dilatation of the arterioles, not of the face only, but, as Romberg shows, it extends over the genitals and thighs. The more the nervous system in men approaches the feminine type, the more liable is the owner to palpitation, while some men's nervous systems seem as if such disturbance were almost impossible.

Prognosis.—The prognosis of palpitation itself is not serious, but it may indicate a grave condition. But palpitation of a violent character, such as obtrudes itself forcibly on the patient's attention, is more decidedly the characteristic of nervous disorders of the heart than structural lesions. In people feeling well ordinarily, violent palpitation is much more probably due to arteriole spasm than cardiac debility. Palpitation is not so especially a characteristic of organic disease, and when it then occurs is rarely so violent, and is induced by effort rather than by emotion. It is easily induced in cardiac dilatation, and is then persistent, that is, it is not more easily induced by effort at one time than another. Some elderly people rather like to show to their medical attendant how easily palpitation is induced by a few quick steps across the floor. But this is not nervous palpitation, and must not be so considered; it is palpitation otherwise induced. In advanced disease of the heart, palpitation is almost a good symptom, as showing that the heart can still palpitate; and in chronic irregularity, palpitation on effort is not nearly so serious a matter as when intermittency or syncope is so induced.

True nervous palpitation may indicate nervous exhaustion; and its recurrence at shorter intervals, or in greater force, and for more prolonged periods, is not a symptom to be overlooked. It may indicate mental strain of a nervous system tried from its psychical and not its physical side. It may indicate in celibate females that the nervous system is beginning to suffer from an enforced involuntary continence. Its prognostic import is not so much with itself as with the condition to which it belongs, or in which it takes its origin. Peter Frank is a well-known case of palpitation induced by arduous study of heart disease, and the action of the heart may be increased in rapidity or in force by voluntary attention to it. Where nervous palpitation alarms the patient, it is more likely to perpetuate itself.

Treatment.—The treatment of nervous palpitation, like that of angina pectoris, has two divisions—the treatment of the paroxysm, and the general treatment during the intervals. The treatment of the attack consists of placing the

patient on a couch, or in bed, avoidance of all appearance of alarm, and a suppression of exuberant sympathy, with the use of eau de Cologne or sal-volatile. It is questionable how far musk or other animal scents act on the emotional nature through the sense of smell, as Laycock suggests, though it is difficult to see any other physical action, but probably they act by diverting the attention. If connected with a hysteric paroxysm, C. J. Hare's suggestion of holding the nose till a deep inspiration is induced, may be put in practice. The action of a deep inspiration is inexplicable, but its efficacy is wonderful in arresting a hysteric paroxysm. When palpitation is induced by sudden effort, rest, quiet, and a diffusible stimulant are indicated, with a few drops of tincture of belladonna or digitalis.

The treatment of nervous palpitation during the interval is a somewhat difficult subject. It means the treatment of the condition on which it depends, and that is a wide subject. A brief bird's eye view is all that is admissible here. Where it is due to a finely strung and susceptible nervous temperament, of course no treatment can affect that; but quiet, mental or bodily, avoidance of all exciting pursuits, which would tend to make the system more hyperæsthetic, are indicated, while any trivial visceral disturbance must be attended to at once, for a system of that kind will not right itself without suffering. Where it is associated with in-door occupations, fresh air and exercise are imperatively dictated. Cold water sponging or baths are ever of service. The excitement of modern fiction is not without an effect on the emotional nature of its votaries, who become as abandoned to this form of intemperance as others are to the use or abuse of other stimulants. The enthralling plot, which the victim to novel reading demands, is allied to the demand for brandy in the toper; slighter stimulants are inefficient and powerless. This excitement, occasioned by plot or story, affects the emotional nature, so closely allied to the sympathetic or ganglionic system, by increasing its susceptibility. Here removal from a circulating library is a necessary step, and exercise, other occupations, and rational mental pabulum, the interest attracted in some other direction, are necessary.

More hopeless still is the case where the mental activity takes a theological, or so-called religious, direction. Here a conviction of higher duties than those connected with the natural man presents an almost insuperable barrier to the adoption of medical advice. The conventual seclusion of the Romish Church had at least one good, in the prevention of another generation of beings with this highly developed emotional nature so destructive of their own comfort. The progress of civilisation, and its effect in heightening the susceptibility of the nervous system, both cerebral and ganglionic, bears heavily on our unmarried or single sisters. The impulses or promptings of the affectional nature become, as Maudsley well has shown, stimulated by a life of constrained celibacy, and the denial of their gratification adds to their pertinacity till they become dominant; this reacts upon the circulation within the ovaries, and persistent ovarian congestion converts these promptings into almost omnipresence, and these occasional longings become developed into physiological necessities. *Homo sum! et nihil humani a me alienum puto!* And the life which is before many of the weaker sex cannot but tend to induce them to be frail. We can find charity for soldiers, where their military regulations do not permit matrimony. Can we not be at least as charitable to those of the female sex, who are quite as stringently compelled to be continent, with even less to occupy their attention? With matrimony as almost the sole future presented to her hopes, a spinster's thoughts are necessarily diverted in an erotic direction; nor can we wonder if that affects her susceptible nervous system. Many cases of nervous palpitation, as well as hysteric attacks, are improved, and even cured, when the cares of maternity are added to the caresses of a husband, and the emotions get settled down into healthy channels when the aspirations have attained their gratification. "The treatment of hysteria does not consist in the administration of nauseous gums, but in proper mental, social, and moral management."—(Russell Reynolds, in "System of Medicine.")

The medical treatment consists of cold-water baths, the use of the bidet, the unloading of all pelvic congestion, and

especial attention should be paid to the condition of the rectum, too apt to be neglected. When the nervous system is more than ordinarily excited, camphor and bromide of potassium are indicated. Disturbed rest is better met by early rising, active exercise, and light suppers, than by opiates or other narcotics, or even by morning slumber. While in those common cases of anæmia and chlorosis arsenic and iron, quinine or strychnine, with cod-liver oil, and a nutritious diet, are of service; the combination of hæmatics with nervine tonics is of great importance. All discharges of every kind must be checked as far as possible, as being drains on the system. Belladonna plaisters may be worn with advantage.

Where palpitation is connected with sudden efforts and a weak heart, it is less likely to affect the heart through the sympathetic branches, though this view has the authority of Ludwig, but through the sudden demand on the muscular walls. Here digitalis is of great service, or belladonna, where palpitation is both emotional and from exertion. Avoidance of sudden effort is desirable, and tonics may be indicated. But any temporary measures, though they may give relief for a time, are ineffective as to the general condition, and recurring periods of treatment may not only be necessary, but even desirable.

When palpitation occurs in chronic Bright's disease, it must be treated by the rules to be laid down in the chapter devoted to the combination of heart and kidney disease.

Irritable Heart.—This subject has been especially investigated by Da Costa, of Philadelphia, whose attention was drawn forcibly to it during the American Civil War. By his kindness in sending his papers to the writer, a complete, but abridged view of this affection can be laid before the reader in the language of Da Costa himself:—

"The general clinical history of many of the cases was this:—A man, who had been for some months or longer in active service, would be seized with diarrhœa, annoying, but not severe enough to keep him out of the field; or, attacked with diarrhœa or fever, he re-joined, after a short stay in hospital, his command, and again underwent the fatigues of

a soldier's life. He soon noticed that he could not bear them as formerly; he got out of breath, could not keep up with his comrades, was annoyed with dizziness and palpitation, and with pain in the chest; his accoutrements oppressed him, and all this though he appeared well and healthy. Seeking advice from the surgeon of the regiment it was decided that he was unfit for duty, and he was sent to a hospital, where his persistently quick-acting heart confirmed his story, though he looked like a man in sound condition.

"Any digestive disturbances which might have existed gradually passed away, but the irregularity in the heart remained, and only very slowly did the excited organ return to its natural condition. Or it failed to do so, notwithstanding the use of remedies which control the circulation; thus the case might go on for a long time, and the patient, after being the round of the hospitals, would be discharged, or, as unfit for active duty, placed in the Invalid Corps."

This account clearly indicates that though the provoking cause of the affection is exhaustion of the sympathetic, still there is also a condition allied in nature to Graves's or Basedow's disease, and as intractable to remedies. Some cases seem to have been almost entirely due to exhaustion of the sympathetic, and these readily yielded to treatment and rest, while the others, which partook of the neurosal character of Basedow's disease, were scarcely, if at all, benefited by treatment.

The tendency of the cases was to develop hypertrophy, and in some cases this became marked, but in the majority it was trifling in amount. In one case, a fairly typical one, death from strangulated hernia furnished an opportunity of examining the heart. "The pericardium was healthy; the heart, before being opened, appeared to be of normal size; at its upper portion was a moderate amount of fat; the valves were all healthy; a small clot was entwined in the mitral valve; the auricles were of normal size, and so were the cavities of the ventricles; the muscular structure of these was firm, and the cut surface glistening. While, as already stated, the cavity of the left ventricle did not appear increased, there was a great disproportion between its walls

and those of the right side; these measured less than one-fourth of an inch at some parts, a fraction over one-fourth at the thickest portion; whereas the walls of the left ventricle were nearly seven-eighths of an inch at the thickest part, and varied from a little over one-half to three-fourths of an inch at others. Microscopically examined, the fibres were healthy, some fibres seemed indistinct, but there was neither fatty nor granular degeneration. The nervous filaments of the heart, as far as they were traced out, appeared healthy, but no minute dissection of the heart was made." The physical signs in life were "impulse somewhat extended, but not decidedly abrupt, and of some force; the second sound is very distinct."

Causes.—"In no part of this inquiry is it more difficult to arrive at fixed conclusions, for many causes seem at times to have been combined, and it is scarcely possible, even by the most vigorous analysis, to fix specially upon one." Of 200 selected and well-marked cases, the analysis stands thus:—

Fevers	34	17 per cent.
Diarrhœa	61	30·5 „
Hard field service, particularly excessive marching.	69	38·5 „
Wounds, injuries, rheumatism, scurvy, ordinary duties of soldier life, and doubtful causes	36	18 „
	200	100*

We see from this that hard field service was a large factor in the production of these cases, and nextly diarrhœa, and thirdly fevers. These are depressant causes, and no doubt led largely in the immediate provocation of the malady, but one great factor in the production of this ailment, which is so intimately connected with the ganglionic or emotional nature, is the excitement, and, to some extent, natural anxiety of this war, which manifested a most unusual deadliness. There was much wilful and unnecessary bloodshed, as remarked by military critics, and this, together with the high nervous development of Americans, the social character of the struggle, the obstinate nature of the fighting, as soon as any troops were seasoned, the novel character of many of

* 104 (?).

the military evolutions, all go to make up a total which explains the occurrence of this type of disease in the American war, and to account for its frequency there. The struggle, too, was prolonged over some years, with varying success, which separates this from the wars of 1864, 1866, and 1870-1 in Europe.

Tobacco in its various forms does not appear to have been a producing cause, "some of the worst cases occurring in those who did not use it in any shape." Nor did sexual excess, or the substitutes for it, "produce the disorder, though they predisposed to it, or kept it up." "We find it most readily developed in those previously weak and unaccustomed to fatigue, or subject to readily-quickened circulation. We find it kept up by irksome equipments and other causes, but not generated by them. (Many cases occurred in cavalry and artillery as well as infantry.) In all those cases it was apt to be noticed that, from the onset, the double quick was badly borne."

Symptoms. Palpitation.—This differed in individual cases both in severity and frequency, and considerably, too. Palpitation was usually accompanied by pain over the heart and in the left shoulder; there was often a great deal of distress; the attacks came on variously, often excited by exertion, but at other times coming on at night in bed. As a rule, the patient could not lie on his left side for fear of exciting them. Pain was an almost constant symptom. The chief seat of the pain was the lower part of the precordia, particularly near the apex. There was also hyperæsthesia. The pain was not due to intercostal neuralgia. *Pulse.* This was generally rapid, varying from 100 to 140. In character it was small, and easily compressible. The pulse was always greatly and rapidly influenced by position, varying from 108 when standing, to 80 when lying down, or in another case from 120 to 84. Respiration was embarrassed, and shortness of breath complained of, yet, notwithstanding all the signs of dyspnœa, it was astonishing that the respiration was so little hurried, as for instance, pulse 124, respirations 25; pulse 146, respirations 26. Nervous symptoms were complained of, especially headache, giddiness, and disturbed sleep. The

sleep was disturbed by jerking, or by unpleasant dreams. Digestive disorders were frequent.

Inordinate sweating of the hand was several times complained of, and Da Costa thinks this was due to disorder of the sympathic, and a modification of the general excessive perspiration.

Physical Signs. Impulse.—"This was almost always extended, yet not correspondingly forcible; rather it is abrupt or jerky, and quick." There is commonly some hypertrophy with its characteristic signs, but rarely dilatation. Murmurs, obscuring or replacing the cardiac sounds are not, as a rule, present: when occurring, they are usually systolic and heard at the apex, thus differing from the aortic murmur of anæmia.

Diagnosis.—This is usually not very difficult, and is founded on an aggregation of the signs and symptoms, given above, along with the history. The irritable heart differs from dilatation in the absence of extended dulness and subjective symptoms. It appears that it may most readily be mistaken for phthisis, unlikely as it appears at first sight. There is irritative cough, with expectoration, and often hæmoptysis, especially after exertion. "But the aspect of the patient, the pain in the precordial region, the attacks of palpitation, and absence of the physical signs of tubercle, furnish the distinctive traits."

It is sometimes feigned by tying a tight bandage round the lower part of the chest and upper part of the abdomen. The suspected patient must be stripped and told to lie down, when the rapidity of pulse falls, but does not mount again on his resuming the erect posture. The impostor often overlooks the characteristic cardiac pain.

Prognosis and Treatment.—The prognosis varies much apparently with the malady itself. When indicating rather exhaustion of the sympathetic by hard marching and prolonged excitement, it seems fairly amenable to treatment, especially when combined with rest.

But in other cases, which seem rather allied to Basedow's disease, and to indicate a neurosis rather than any simple exhaustion of the sympathetic, the case is decidedly dif-

ferent, and the prognosis resembles that of Basedow's disease.

Da Costa says, speaking generally, "The treatment is never a short one; and the question arises, would it not be better for the Government at once to discharge these heart cases?" In answer, he thinks it not desirable, as leading to encourage heart affections (functional) among soldiers in war times, and prefers placing these patients in the veteran reserve, or to do mere routine duties, not entailing fatigue and excitement, to their unconditional discharge, which, he thinks, would have a "demoralising effect." Having decided upon retaining them in the service, the next question very naturally is that of treatment. The first point to be attended to is rest, ever of so much importance in the treatment of heart affections. The great difficulty is simply to get rest in these cases, where it is almost out of question from the nature of things. When practicable, it is of the highest importance. Nextly, as to remedies. Digitalis and its active principle digitaline were employed; there was no difference in their utility, "and both had more influence on the cardiac disorder than any other drug which was resorted to." Then came veratrium viride and belladonna, both of which were useful. Belladonna seemed to exercise great control over the element of irregularity, and often advantageously preceded the administration of digitalis and iron. This combination was most useful in cases of debility, combined with greatly increased rapidity of pulse. In other cases, aconite seemed more desirable. "On purely irritable hearts it had very little effect, nay, repeatedly it was noticed that the impulse became more frequent under its use, and even more abrupt."

"But it was, after all, in cases of decided increase in the organ, in cases of hypertrophy, that aconite most showed its influence, he goes on to say, after mentioning its influence in apparently arresting organic changes. As to opium, it appeared to exercise some quieting action on the heart, but is not likely to be of much service ordinarily, so far as its exhibition for concurrent affections, as dysentery, would warrant conclusions. Hypodermic injections of morphia were often

resorted to for the relief of the cardiac pain, and almost invariably, at least for the time being, accomplished the desired object."

Strychnine, quinine, iron, and zinc were all serviceable in their place, and especially after the active irritability had been allayed. "Great care was taken with the men during convalescence. They were mostly placed on guard duty, or other light duty, some still continuing treatment in a modified degree, others not; and were ordered up for examination at stated intervals." Finally, it may be remarked that the treatment appears to the writer to have been conducted on two principles, during the first part of the treatment, viz., to give either agents increasing vaso-motor action and arterial tension, or agents lowering both. That in certain cases digitalis, belladonna, veratrium viride, or strychnine were most beneficial, and that in these cases there was debility and low arterial tension. In other cases with forcible action and tendency to hypertrophy, aconite was more useful. There was an evident antagonism existing, and cases benefited by one plan, usually, were aggravated by the other. In the first series of cases an agent which increased the tone of the cardiac contractions, while acting also on the peripheral muscular portion of the circulatory system, was indicated, in the others an agent which lowered arterial tension, acting on heart and arterioles by lessening their contractibility, was rather of service.

The inseparability of action on the central and peripheral portions of the circulation, *i.e.*, of exciting increased contractility in the central muscular mass, the heart, without a similar action on the peripheral muscular distribution, and *vice versâ*, explains why good effects should have been attained from agents so antagonistic as digitalis and aconite; though, it must be borne in mind, not in the same cases.

The functional affection of "initable heart" as described by Da Costa is quite dissevered from those diseases of the heart in soldiers described by Dr. Myers in his Alexandra Prize Essay, which apparently arise alike in peace and war; and, though unquestionably connected with exertion, are specially due to the tight jackets and padded waistcoats

which seem deemed indispensable to the British soldier, and by which his health, as well as his comfort, is sacrificed to some peculiar æsthetic views as to the ideal physique of a soldier.

Sub-Paralysis of the Heart.—This is an affection in many respects allied to nervous palpitation, including a muscular counterfeit. It is characterised by a feeble, compressible pulse, not unnaturally fast, it may even be slow, with a tendency to irregularity, or to intermit. The heart's action is in accordance with this; the sounds are low, and the impulse feeble. At other times there is great rapidity and feebleness in the pulse, as seen in some cases of delirium tremens, when the delirium is due to anæmia, and the cases yield rapidly to the digitalis.

Sub-paralysis of the heart is an affection which needs more attention being paid to it, and needs clearing up. There seems three conditions under which it occurs, namely, exhaustion of the sympathetic, affections of the vagus, and muscular exhaustion.

Exhaustion of the sympathetic is the most common of all causes, and is variously induced. Excessive tobacco smoking, though borne by some with impunity, is very injurious to others, and it is well known that many men can smoke light tobacco with impunity, who are soon compelled to give up strong tobacco. The pulse is readily affected by strong tobacco, and is then quick, feeble, and tending to be irregular. Exhaustion from excessive stimulants, and from excessive strain on the nerve force, and consequent imperfect supply of the sympathetic nerve centres which preside over visceral involuntary actions, including the heart, is injurious, however produced. The defective action of the heart imperfectly supplies the cerebro spinal centres with blood, less nerve force is evolved, and the sympathetic is imperfectly supplied, and the condition perpetuated. This drain may be variously induced, and one case which came under my notice in Vienna is instructive. The gentleman possessed a physique of more than ordinary vigour, and the muscular or acting force was good, and also the vital forces under exertion. But it so happened that he was also given to sexual indulgence, and

this affected his pulsations markedly. The heart's action was feeble and the pulse small, normally fast, and very compressible. This was more marked after indulgence, but became somewhat chronic. Once previously, under similar circumstances, the same state had been induced. At last, getting uneasy about it, he went off for a walking tour, somewhat doubtful about his power to sustain exertion. But, to his astonishment, he soon got a good pulse, was more equal even to exertion, mountain climbing gave no inconvenience, every symptom of cardiac debility passed away, until, during his Viennese residence, the old habits were resumed, and with them came back the old feeble condition of heart and pulse, with exacerbations too markedly connected with his indiscretions to leave any doubt about their standing to each other in the relation of cause and effect.

The condition of sub-paralysis of the heart from exhaustion of the ganglionic system is a subject of more interest than it has excited, and the imperfect contraction of the heart, due to this ganglionic failure, is of interest, practically, as well as bearing on the subject of cardiac innervation.

Sub-paralysis of the heart is an affection which must not be connected with abnormal slowness of the pulse. Some persons have an abnormally slow pulse, without any disease or inability. One case was reported to me in Berlin, where a gentleman had been rejected for military service on this account, though quite healthy and well. His pulse is ordinarily 40, and when ill may mount up to 80, showing an increase in number of beats quite normal enough, and demonstrating that there is no disorder, but merely what, in lieu of more knowledge, we must denominate a peculiarity. Cases of abnormal slowness occurring in sickness are not unknown, and in one whole family in the north of England this occurs. The pulse falls to 35 or 40 during illness, but this does not appear a source of danger, and the condition during health is that of robustness, and the life of average duration.

Sub-paralysis cordis may be produced by causes acting through the inhibitory action of the vagus, and one noted case, where this was diagnosed during life, is worth relating. It occurred in the Krankenhaus of Vienna, and was seen by

Heine and Skoda. The heart stood still for five or six beats. During this time the patient's appearance "told that something terrible was going on within him;" he suffered mental as well as bodily agony. He ultimately lost the power of speech, and had paralysis of all his extremities. The diagnosis was tumour of the vagus, with hypertrophy of the cervical portion of the spinal cord. Skoda's diagnosis was almost unerring, when he ventured one; and Rokitansky found the lesions diagnosed, the tumour in the vagus being about the size of a cherry, and embracing the nerve. This case is almost unique, and Romberg, in his work on the Diseases of the Nervous System, does not give another (vol. ii, p. 340).

Arrest of the heart's action by will through the vagi is a common practice with Indian jugglers, and one Indian officer acquired the art, and ultimately fell a victim to a prolonged effort, from which he never recovered. In shock, or syncope from mental emotion, it is perhaps not easy to say how far impressions from the brain act through the vagi in producing cardiac syncope, but it is not probable that this is the usual causation. Shock is described by Romberg as a "paralysis of the sympathetic," and it is often associated with causes which remove it from influence through the vagi. Collapse is a similar arrest of action, acting through the sympathetic rather than the vagus.

Muscular Sub-Paralysis.—This affection was described by earlier writers as acute distension or engorgement of the heart, that is, a condition of ventricular fulness to the verge of paralysis. That, in diseased hearts, this may proceed to final cessation in diastole, is well known as a common cause of sudden death, but it is a not uncommon condition in healthy hearts. It is especially liable to occur in the right ventricle on effort, for when effort is protracted as well as laborious, the impeded circulation, due to the accumulation of non-oxygenised blood in the lungs, and the distension of the right side of the heart, possibly aided by the presence of so large a quantity of blood charged with carbonic acid, necessitate cessation of all effort from dyspnœa, and a distressing sense of distention and pulsation in the heart. Cessation of

effort gives relief, but the resumption of effort reprovokes the unpleasant symptoms. They are apt to return, or even to persist in a chronic form of cardiac asthma. Dr. Clifford Allbutt has given a description, with his usual felicity, of an attack which occurred to himself.* After a long day's Alpine walking, a further ascent was attempted. This new effort produced suddenly "a strange and peculiar *besoin de respirer*, accompanied by a very distressing sense of distension, and pulsation in the epigastrium. On placing my hand over my heart, I felt a labouring diffused beat all over the epigastrium," he goes on to say. Rest gave relief; but climbing brought it back. After reaching level ground all was well; but at about three in the morning he was awakened suddenly with a return of the attack. Dr. Allbutt may be congratulated that that morning attack was the last he knew of his over-distension of the right ventricle, as several cases of its remaining for a considerable time have come under my notice. The prolonged strain, or frequently repeated strain, on the right ventricle produces partial paralysis and loss of resisting power. Thus swimmers and divers find that they can "train off" as well as "train on." To "train on" means improvement in wind, and to "train off," the opposite. The right ventricle is then overstrained, and becomes less equal to demand on it. In the diving mammals the right ventricle is comparatively much stronger than in other mammals, and supported by trabeculæ, of which the columnæ carneæ are the homologues.† In two very marked cases of cardiac asthma in healthy young men, this prolonged effort, after nature's admonitions to desist have been disregarded by "pluck," was the cause in both—in one after prolonged gymnastic feats in a competition, where hernia resulted at the same time from the efforts made, and in the other, running long after marked uncomfortable feelings were present, but disregarded in favour of other even more strongly impelling motives. The same thing occurs in

* "The Effect of Overwork and Strain on the Heart and Great Blood-vessels." 1871.

† To those interested in this subject, the writer can refer them a paper by him in the "Edin. Monthly Med. Journ.," Dec., 1870, under the somewhat clumsy title of "Cardiac Distensibility, Distension, and Dilatation."

horses that are broken-winded from a hard ride or hunt, and a winter or summer out at grass is a common cure. It is not to be supposed that there is any curative property about grass, as compared to its dried condition, hay; it is the rest from exertion, except such as is voluntary and can be desisted from at the horse's will, and not his rider's, that permits of recovery. The horse usually drops, as does the soldier out of the ranks, when "on the march," from cerebral anæmia due to this condition, or rather to deprival of arterial blood. The distension of the right ventricle, and the general venous congestion, connected with prolonged muscular effort, as we have seen in a previous chapter, lead to active venous congestion within the encephalon, while the right ventricle, transmitting only a small quantity of blood over to the left ventricle through the gorged lungs, there is only a small amount of oxygenised arterial blood sent to the brain, and the brain falters for want of its arterial blood just as we have seen it does in chronic heart disease.

A curious account of the effect of strain, of over-exertion, was related to me by my friend Dr. Brunson, of New York, in Vienna. When doing a pedestrian tour in the Tyrol, after a long and arduous ascent of the most perilous character, he felt his head swim when on a giddy height; being a man of iron nerves, he kept complete command over himself during the perilous feats requisite ere he could again descend from the peak. On discussing the subject with his guides, which his command of the language enabled him to do freely, he found that a total loss of self-command and of nerve was not by any means uncommon on the peaks and glaciers, and the victim had often to be literally borne down as best might be by the guides. These guides had also observed that severe and long-sustained efforts had usually preceded these attacks, or the sufferers had been previously out of sorts, or not in good condition. Here there can exist no doubt that there was cerebral anæmia from an exhausted and imperfectly-contracting heart, due to a condition of general exhaustion of the sympathetic. Many of the sufferers had done many ascents and feats of climbing previously with-

out any such feeling. Interesting as would have been the account of Dr. Allbutt's experience had his attack seized him on a peak or pass, and to be regretted for the sake of science, as it will not often happen that so capable an observer is in such a physical condition among the Alps, and even more interesting if there were also this cerebral anæmia (though I am afraid he is too cool to have exhibited this phenomenon very markedly), it is possible the experience would not have been pleasant personally, however scientifically interesting.

This condition of over-distension of the right ventricle is quite common in chronic disease of the heart, and also in the curious attacks of dyspnœa in chronic Bright's disease, where spasm of the pulmonary arterioles, excited by the impure blood, is the probable cause of the right-side failure, venous congestion, cyanosis, and small arterial pulse connected with this condition. Dr. Peacock, in his Croonian Lectures, 1865, thinks that the impure air of the mines has something to do with the dilatation of the heart in Cornish miners, as well as the exertion required, and with this opinion I quite agree, as the imperfect oxygenation and highly venous condition of blood must affect the right ventricle and tend to paralyse it; as we know that Cyon produced paralysis of the heart from serum charged with carbonic acid, and that the contractions returned when the carbonic acid was shut off, and ceased on its being again added (Sydenham Society's Year Book, 1867-68). This sub-paralysis of the right ventricle may become something more, and is a not unusual cause of death in acute zymotic disease and affections of the respiratory organs. Here the pulse mounts higher and higher, becomes irregular, then more irregular and intermits. and then finally ceases in diastole, and on the *post mortem* the right ventricle is found gorged with blood, while the left ventricle is empty and contracted. It is obvious here that the mounting and irregularity were connected with the right ventricle, and not the left, for the left had no obstacle to encounter, but must keep time with the right in consequence of the fibres common to both, and so

transmit to the arterial system the character of the contractions of the right ventricle.

Treatment.—This depends upon the nature of the complaint with which this sub-paralysis is associated, on which the diagnosis and prognosis also depend, for in some cases it is only a neurosal or otherwise passing affection, in others a grave chronic condition, and in others again the precursor of death. Removal of the exciting causes, where practicable, is, of course, demanded, with the administration of stimulants and perfect quiet insisted on for a time, in the acute attacks; while in the more chronic conditions of exhaustion of the sympathetic good food and altered habits are desirable; and in muscular debility of the heart walls digitalis or belladonna, with iron, quinine, or strychnine, are indicated. In cases, like Heine's, of tumour of the vagus, nothing curative can be done, and palliative treatment is all that is feasible, though it is not likely such an extremely clever diagnosis can often be made. In those cases of acute over-distension connected with muscular exertion, care for the future for those who have suffered, and advice to those who may suffer, as to avoidance of prolonged exertion, especially when not in good condition, are available; while the more persistent chronic condition is to be met by agents which induce more perfect ventricular contraction, and avoidance of aggravation by new efforts is to be recommended.

Basedow's or Graves' Disease. Exophthalmic Goitre.—This affection has had great attention paid to it by numerous intellects of the highest order, and is connected with the names of Basedow, Graves, Stokes, Begbie, Aran, Trousseau, Charcot, Remak, von Graefe, Oppolzer, Traube, Virchow, and von Recklinghausen, &c. It still remains, however, without any positive pathology, and its ætiology is yet shrouded in mystery. Its prominent points consist of a curious union of three apparently totally separate affections, viz., heart disturbance, enlargement of the thyroid gland, and unusual fulness or prominence of the eyeballs. To these may be added a decidedly emotional temperament. It may be well to consider each division in turn in relation to the

changes observed in them, as the phrase "the pathological anatomy" is scarcely quite applicable.

The Heart.—The symptoms associated with the heart are decided tumultuous action, extreme rapidity of pulse, amounting to 160 or even 200 beats per minute, while the heart seems thrown bodily against the thoracic parietes at each systole, and the impulse is diffused over a large area. There is usually some enlargement of the heart, and increased dulness, and the heart much resembles in every respect a dilated and hypertrophied heart under excitement. There may also be valvular disease, but it is not characteristic; a systolic murmur may be heard at the apex, and a thrill be felt, but this is liable to occur from disordered action of the musculi papillares. As well as the excited action about the heart, there is also abdominal pulsation, and the carotids are full, and give a thrill. The radial pulse is usually small, and not in proportion to the heart's action.

The Thyroid.—The thyroid gland is enlarged, may vary in bulk, and has not the firm feel of bronchocele, but rather an elastic feel, and pulsates. This is due to a dilated condition of the branches of the thyroid artery. Under excitement, the thyroid is enlarged even more and the sensation of pulsation is increased. There may be a thrill or even a murmur, and the right lobe is sometimes larger than the left.

The Eyes.—The expression of the eye varies, and when it is only slightly prominent, it appears little more than a full, large eye, giving an interesting expression to the features. When the eyeball is more prominent, it gives a wild, scared look to the person, somewhat resembling the look of a hunted animal. In some cases the eyelids are unable to close over the eye, and this is seen in sleep markedly, the cornea is then imperfectly covered, and this, together with the tension, leads to various changes in the cornea. Thus, soon it becomes catarrhal, or ulcerates, or becomes infiltrated with pus. The upper eyelid is swollen with blue veins in it. Von Graefe regarded a retraction of the upper eyelid with a downward cast of the eye itself as pathognomonic of this affection. The pupil is normal, and the ophthalmoscopic signs

negative. There is sometimes neuralgia of the ophthalmic branch of the fifth nerve.

The Temperament.—The temperament is emotional, susceptible, and sensitive. This leads to mental disturbances, and the patient is easily distressed, apt to attach more importance to little matters than they really deserve, and, if a married woman, is liable to frequently make quarrels with her husband. When in Leeds Public Dispensary, the writer always asked if they felt often inclined to quarrel with, or scold their husband, and after a crimson blush an affirmative answer was always given. This inclination to have little quarrels and arguments was uncontrollable, and to some extent involuntary. The patients are usually of a somewhat refined temperament, and these mental ebullitions distressed them much after. There is a great approach to the hysterical temperament in these patients. The very fact of having a greater inclination to be angry with their husbands showed the emotional character of the affection, and in all probability the real cause was some imagined indifference, or trifling neglect, brooded over and magnified by the peculiar mental condition which accompanies this disorder, and is as much a disease and removed from the patient's will, or control, as the enlargement of the thyroid or the protrusion of the eyeballs. There is also usually present catamenial derangement, and the menses are scanty or irregular, in others profuse, and in some patients leucorrhœa accompanied the menstrual disorders.

Sex.—Women are very much more liable to this affection than men, and from their emotional temperament this is not unlikely. Von Graefe found only one man to every seven women among patients affected with this complaint.

Age.—The period of life to which exophthalmic goitre essentially belongs, is that betwixt puberty and the menopause. It may be found over either limit, but rarely. In men, adult life is also the period during which this affection manifests itself.

Nature of the Affection.—It is now generally admitted that this affection is a disease of the sympathetic or ganglionic nervous system, and that the vaso-motor, or trophic, nerves are the part most especially affected. There is disturbance

of arterial and arteriole calibre by disturbed vaso-motor action, here contraction and there dilatation. Thus the arteries of the thyroid are dilated and enlarged until the thyroid is almost an erectile tumour. So the prominence of the eyeballs is due to a similar condition of the blood-vessels behind the eye, with a development of fat-cells. The condition of the heart Niemeyer states confidently to be due to a paralysed condition of the vaso-motor nerves of the coronary circulation. The production of the disturbance in the heart is not in all probability simple, and in addition to disordered innervation of the heart, there is also obstruction to the flow offered by this irregularity in the calibre of the vessels, and thus the heart walls become dilated or hypertrophied, or both. For dilatations of a tube, no less than contractions, increase the difficulty of flow through it, and are obstructive. The vaso-motor nerves are connected with the ganglionic system, and so are the cardiac ganglia, on which depends the heart's contractility, and there is reason to hold that their action is somewhat diminished, and that the increase in size of the ventricular chambers is partly the result of disordered innervation.

The scalpel has not yet revealed the pathology of this affection, nor has the microscope been any more successful. It has been thought that an enlargement and redness of the inferior cervical ganglion, with sparseness, diminished size, and fatty development among the ganglion cells, would account for it; but any pathology which can be regarded as causative, or connected with this affection, other than as results of it, is yet to be discovered.

In the heart fatty or amyloid changes are found, and often valvular lesions, while the aorta and larger arteries may become atheromatous; the thyroid arteries may become almost aneurysmal and dilated; while a development of connective tissue in the gland may lead in time to its becoming smaller and harder; while in the eye, the vessels behind may become diseased, the muscles of the eye degenerate, and the lachrymal gland atrophied, probably from the same cause as the thyroid.

Cerebral hæmorrhage has also been found.

History and Duration.—The affection comes on from disturbing causes, physical or mental in a manner not yet explicable; and the favourite subjects are fair women with blonde hair, blue or hazel eyes, with the temperament termed by Laycock nervous, that is, there is high nervous susceptibility amounting to hyperæsthesia.

The duration may be from a brief period of weeks to years, and usually it does not cease till the menopause. Nor in many cases is its duration shortened by remedies; a certain alleviation may result from their administration, but the temperament on which it depends is unalterable.

Prognosis and Terminations.—The prognosis as to time is certainly not good, but as to life it is much better. Death may result from intercurrent affections, some of which may be traceable to the affection; but others are not so associated. The affections more distinctly traceable to the disease itself are heart diseases with all the results which spring from its failure, whether connected with valvular failure or disease of the walls, cerebral hæmorrhage, or thickening of the meninges. In some cases it seems connected with syphilis, and the constitutional consequences which result therefrom cause death; but it can scarcely be said that these results followed from the exophthalmic goitre, which was itself merely a consequence of the general cachexia, and no more responsible for the results than could be the exanthemata, which also resulted from the acquired disease. In a disease so liable from its nature to be chronic, death must not uncommonly occur during its existence, but these deaths may not in any way be due to it, but quite unconnected with it, and therefore in reading statistics of it, the deaths must not all count against the disorder. It is not a fatal disorder in itself, but death may result from it, but more often occurs during it. Of 56 cases, von Dusch had 14 complete recoveries; great improvement in 26; no improvement in 4; 7 died; and in 5 the result is unknown. Von Graefe had 12 per cent. of deaths, 20 per cent. recoveries, 30 per cent. great improvement, and 38 per cent. unknown. It is neither a fatal nor a curable disease, as a rule; and it is difficult to comprehend what condition remained after marked improvement or re-

covery, as it is to understand how far the deaths were connected with the affection.

Diagnosis.—This is frequently easy enough, the combination of heart disturbance, with goitre and exopthalmos, are usually so marked as to make the diagnosis one of no difficulty. But at other times one of these three may be wanting, or even one only may be present, and then it is not so easy to make a diagnosis. It is very desirable, however, to do so, as from the known intractable character of the ailment it affects the prognosis. Palpitation when associated with this neurosis of the sympathetic, is much less tractable than when connected with temporary failure, or the opposite condition of vaso-motor disturbance with spasm of the arterioles, occurring during limited periods. So this thyroid enlargement is not to be cured as many are by iodine ointment; almost a specific in my experience of bronchocele common in my native village.

The absence of one of the different component parts often obscures the diagnosis, but in cases of doubt the mental or emotional disturbances will often aid in forming a diagnosis, and where the peculiar temperament is discovered, so allied to the hysterical temperament, a clue to a diagnosis, as well as a prognosis, is furnished. The temperament is incurable, though some of its manifestations may be relieved. This mental disturbance may itself be a source of danger to life, as in self-destruction, or the mental balance may be so disturbed as to result in mania.

In addition to the mental condition there is another aid to diagnosis, the "tache cerebrale" of Trousseau. This is the production of bright red flecks or spots on the skin of the head by slight irritation of the nail. This is a transitory and localised paralysis of the vaso-motor nerves of the minute vessels of the skin immediately under the surface irritated. This phenomenon is precisely that of "blushing," which is a momentary general vaso-motor paralysis, only it is localised. Trousseau has deemed this a pathognomonic sign of this "neurosis of the sympathetic" under consideration. This, however, it is not; but in establishing a diagnosis it must not be forgotten.

Treatment.—This is not a very satisfactory matter. Digitalis is recommended by Trousseau, Oppolzer, and others, and would seem to meet the condition of dilated heart and dilated arterioles most exactly from its action, but whether this condition of the sympathetic is unfavourable to the manifestation of impressions on it, or these impressions are not produced in its altered condition, or what, it is not easy to say, but certainly it has not appeared to me, at least, to have much effect on this condition, in which it resembled all other specific treatment of it. Belladonna is also a remedy of repute. Iron in anæmia is good; but Trousseau is not in favour of it generally. Preparations of iodine have been praised, and the combination of it with iron, as in Blancard's pills, is strongly advocated by Oppolzer and others.

Remak recommends the use of the constant stream, and the application of the magnetic current is consistent with our knowledge of its action on involuntary muscles and organic nerves. Where there is great cerebral excitement and mental manifestations, bromide of potassium or ergot of rye might be tried.

But the treatment resolves itself into the removal of all causes which might render a susceptible emotional system more hyperæsthetic. Therefore psychical as well as physical treatment must be adopted. The patient must, if possible, be taught self-control, for her own comfort, all must be avoided likely to upset her, excitement or bodily exertion. Where there are catamenial derangements they must be cured or amended, leucorrhœa must be checked, and uterine affections subjected to their appropriate treatment. If the condition or diathesis is itself beyond the reach of remedies, the more need for relieving or removing what is within our reach.

Choreic Palpitation—The condition of the heart in chorea is yet unexplained: it has, however, many points of resemblance to the above disorder. Here the whole heart seems also thrown violently against the anterior wall of the thorax, which almost vibrates from the shock. In the absence of any pathology to chorea, it is difficult to speculate even upon

the immediate cause of the heart disturbance. This is known, that the chorea of childhood and puberty, where the nervous system seems somewhat to lag in its growth, is a condition curable in time by nature or art, and the condition of the heart improves with the nervine tonics, &c., which usually improve the general condition, among which stand foremost arsenic and iron, and even more their combination.

Hyperæsthesia of the Heart.—This is a condition the opposite of sub-paralysis, and consists of an uncommonly quick pulse, quite compatible with perfectly good health. As, however, a rise in the rapidity of the pulse is among the prodromata of many affections, it is well to be guarded in an opinion as to the rapidity of the pulse and its causation, until the case has been some time under your own, or some other observation, which can be relied upon. This condition is a mere disturbance of nerve balance, a hyperæsthesia of the sympathetic, where the heart seems to be excited to contraction by very slight distension, and an unusually rapid pulse results. How far it falls in disease or during an acute disorder is not known yet, from want of the necessary observations; and it is with the hope of attracting attention to this matter that the writer makes this statement, for in the exactly opposite condition of delayed or abnormally slow pulse this may occur.

CHAPTER XIII.

Combined Heart and Kidney Disease.

Part I. 1*st Stage.* From Kidney Disease to Heart Changes: Effect of Imperfect Eliminations on Arterioles—Hypertrophy of their Muscular Walls—Effect of this on the Left Ventricle—Hypertrophy—Arterial Distension and its Results—Symptoms—Physical and Psychical—Grouping of Symptoms—Anæmia—Compensating Actions—Diagnosis—Prognosis and Progress.

Part II. 2*nd Stage.* Heart Failure: Effect on the Venous System—Secondary Affection of the Kidneys—Symptoms (old and new blended)—Diagnosis—Prognosis and Terminations. Treatment of 1st Stage and 2nd Stage:

Kidney Disease the Consequence, and not the Cause of Heart Disease—Pathology—and Therapeutical Indications.

The combination of disease of the heart with disease of the kidneys has been a subject of considerable professional interest since the days of Dr. Bright, whose name is so inseparably connected with research on chronic renal disease. It was first observed that hypertrophy of the left ventricle was often found in cases of chronic kidney disease without any apparent cause. Bright thought that the blood, altered chemically by imperfect depuration, flowed with more difficulty along the arteries Mr. James, of Exeter, had previously, so far back as 1817, pointed out that obstruction in the small arteries caused a hindrance to the flow of blood which, in its turn, led to hypertrophy of the left ventricle. His view was that which was subsequently announced by Bright, viz., "the altered quality of blood might so affect

the minute and capillary circulation as to render greater action necessary to force the blood through the distant subdivisions of the vascular system." In 1855, Traube published some writings on the connection (Zusammenhang) betwixt chronic heart and kidney disease; but, unfortunately, he has not published his more recent ideas, which are not quite what he once held. The idea of the zusammenhang with which his name is associated, is this 1st contraction of kidney and impeded flow through it; 2nd, obstructed arterial flow in consequence of this; 3rd, hypertrophy of the heart. Traube himself is aware that the muscular tunic of the arterioles is thickened by hyperplasia in chronic Bright's disease.

This most interesting discovery, which has done more to unravel the previously mysterious connection betwixt chronic kidney disease and hypertrophy of the left ventricle, was first announced by Professor Geo. Johnson, of King's College, in the 33rd volume of the Medico-Chirurgical Transactions. After further observation, Professor Johnson found that this thickening was always present. "In fact, it occurs with the constancy of a physical law," he says in 1870 (Brit. Med. Jour., April 16th). Professor Johnson regards this hypertrophy of the muscular tunic of the arterioles as due to increased contraction of the small arteries, excited by the abnormal quality of the circulating blood. This in its turn excited hypertrophy of the left ventricle from the obstruction offered to the blood stream by these contracted arterioles, the condition being general and "about equal in all the tissues, the kidney excepted in some cases; while in other cases it seems to be greater in some tissues than in others." Johnson's position is this, as to the relationship of the diseases in heart and kidneys. 1st. Disease of the kidney, and imperfect depuration of blood. 2nd. Arteriole spasm, leading to hypertrophy in their muscular tunics, and obstructed blood-flow. 3rd. Hypertrophy in the left ventricle in consequence of the obstruction to the blood stream. This last view is much more complete than Traube's, which is generally held in Germany, though discarded by Niemeyer, Bamberger, and others. It would scarcely be fair to Professor

Traube to charge him with still holding his old views unmodified, but I have no authority from him to state his present views, which no doubt he will announce when it seems fitting to himself. It is enough for the present purpose that Professor Johnson's views are corroborated by Dr. Quain, Professors Rutherford and Garrod, and Drs. Kelly and Broadbent. When in Vienna I brought Johnson's views under the notice of Professor Stricker, but, on mentioning the matter to him on leaving Vienna, he declined to give any positive opinion, as he alleged his observations were not sufficient to warrant a direct expression of opinion. His views, however, were more favourable then to Johnson's than at first. In attempting to describe this combined condition, the teaching of Johnson will be adopted here.

It must not be forgotten that a form of kidney disease results from heart disease, a general interstitial inflammation, with growth of connective tissue. As the first form of heart disease from kidney disease merges, on heart failure, into the second form of kidney disease resulting from heart failure, and thus the symptoms special to each become blended and so lead to confusion; the subject will be divided into two parts, viz., 1st. The changes from chronic kidney disease to changes in the heart being established; and, 2nd, dating from the time of the setting in of heart failure, the changes which follow from heart failure, and especially the effect upon the kidneys.

Part I.

(*From Kidney Disease to the establishment of Heart Disease.*)

For a better understanding of the circulation through the kidney, we are indebted to Virchow. And as the work of Niemeyer is the best and most widely circulated medium of communication betwixt German pathology and English readers, the views, as announced in Niemeyer's chapter on hyperæmia of the kidney, will be adhered to here. The renal artery has three divisions or branches, first the convolute, passing into the cortical substance of the kidney, and connected with the elimination of water through the thin-walled

glomeruli in the Malpighian bodies; second, the vasa recta connected with the nutritive processes in the kidney, and also the elimination of urine solids; and, third, an intermediate set, both regionally and functionally, which partake of both functions. The water is eliminated by simple pressure on the glomeruli (Bowman), and then in passing down the tubuli uriniferi, filtering from epithelial cell to epithelial cell, becomes more and more charged with urine salts (Beale). The effect of pressure on the glomeruli is increased by the venous capillaries, if they may so be called (for they are merely on the venous side, and do not contain venous blood, but merely blood deprived of so much water), which are unusually narrow and of small calibre. This has two actions, the one to obstruct the flow in the glomeruli, and so produce more pressure and water elimination, and the other to diminish venous pressure in venous congestion.

This diminished bulk of urine is connected with defective arterial tension positively, and not with venous congestion, offering obstruction to the flow. That is when in conditions of impeded circulation, there is falling off in the bulk of urine, it is not that there is venous congestion, but that the veins being fuller of blood the arteries are emptier, and so arterial tension is less, and elimination of water less. When the abdominal aorta is tied below the renal branches, and thus tension on the glomeruli increased, there is largely increased bulk of urine, but without albumen, positively without albumen, or alteration in amount of urine salts, they are in equal amount, but in a higher state of dilution. Urine salts are eliminated from the nutrient branches of the renal artery apparently by a vital action in the epithelial cells. Ligature of a renal vein produced an albuminous condition of urine, with exudation casts and blood corpuscles (Frerichs).

These are important distinctions; as increased arterial tension, which we will see occurs in the first division of our subject, is merely a difference of degree from ligature of the aorta below the renal branches; while venous stagnation, or congestion from heart failure, is a mere difference of degree from ligature of the renal vein.

In chronic Bright's disease, to follow Niemeyer's patho-

logy, there is inflammation of the uriniferous tubule from its Malpighian body downwards, this is followed by atrophy and destruction of it, its cells, and blood vessels, and usually a growth of connective tissue around it. Thus a certain portion of the kidney is destroyed for all functional purposes. There is now a disturbance of balance betwixt the histolytic products of the body and their depurative organ the kidney. The blood is now surcharged by an excess of the products of retrograde tissue-metamorphosis, normally present in it. From this blood-poisoning follows a most interesting series of compensatory changes, and however distinctly compensatory they are, it must never be forgotten that a pathological, and therefore necessarily downward course, is under consideration. Consequently, while compensatory changes are spoken of, it must not be imagined that they are perfectly compensatory, or not morbid actions, and that each compensatory change though conservative in its way, and tending to the preservation of life, and also to some extent of health, has destructive consequences following in its wake, inseparable from itself. Unalloyed good is not to be found in pathological processes any more than anywhere else. This condition of imperfectly depurated blood produces spasm of the arterioles,* which are almost entirely muscular and regulate the blood supply to the tissues, while at the same time they convert the intermittent arterial flow into one continuous equable stream. They are the muscular structures at one end of the circulation, the muscular heart being at the other, and tubes, not entirely but practically elastic, between them. Action goes on back and forward betwixt these two, to some extent antagonistic, muscular terminations of the arterial system, with what effect and result on the elastic tubes betwixt them we shall see. This spasm of the arterioles has one effect which must not be overlooked; by reducing the flow of blood through the tissues generally, it reduces the chemical interchanges, there is less muscular

* Ludwig, Traube, and others say that this is brought about by the effect of retained urine salts on the vaso-motor centres, and have produced arteriole spasm by injection of urine into the blood. This vaso-motor centre has been determined by experiments of Ludwig, von Bezold, Thirry, Cyon, &c.

and other waste, and thus the blood surcharged with waste, and imperfectly depurated, is prevented from further poisoning; that is, the depuration being imperfect, the amount of waste is reduced for the time being, and thus the production of waste products is diminished until the blood is again restored to its normal purity, or impurity. Further contamination is thus averted, and we see that the arteriole spasm is conservative in its way. But this spasm leads to two results; obstruction to the arterial flow generally; and to hypertrophy of the muscular tunics themselves, from frequent spasm.

The obstruction to the blood flow in the arteries is kept up by the thickened tunics (which are distinctly muscular, and not to be confounded with amyloid deposit in them), and leads to changes in the left ventricle. These may be divided into hypertrophy, hypertrophy with dilatation, and dilatation. Sometimes no change is inaugurated in the heart wall, but here the ventricle can empty itself completely, in spite of the spasm. In other cases imperfect ventricular contraction leads to distension, and dilatation arrested by hypertrophy, more or less perfectly, as seen in the chapter on Hypertrophy and Dilatation.*

The existence of this thickening of the muscular tunics of the arterioles in all cases, whether the left ventricle be hypertrophied, normal, or dilated, most effectually disposes of the hypothesis, to whom due I cannot remember, that the arteriole thickening is a result of heart hypertrophy, and compensatory to it. In a great majority of cases the left ventricle is found hypertrophied. Here now we have contracted arterioles, and a hypertrophied heart acting against them. Thus the first pathological conservative action has led to another morbid change. Betwixt these two opposing muscular changes we have increased arterial tension, and this, by increased pressure on the glomeruli of the kidney, leads to the prominent symptom, increased flow of urine. The explanation that the increased flow of urine was due to

* A paper by the writer on the cause of hypertrophy, and showing its probable—to say the least of it—dependence on imperfect emptying of the ventricles, and the effect of that on the trophic nerves, in the *British Medical Journal* (March 2nd, 1872), may also be referred to.

increased pressure on the glomeruli remaining healthy, that is, that the calibre of the renal artery remaining the same, while its peripheral distribution was diminished, there must be more pressure on the glomeruli remaining sound, and thus the bulk of urine increased, has been held to be unsound. There is no difficulty in understanding this: this would merely account for a bulk of urine equal to what existed before the kidney disease. The increased tension, due to the opposing muscular terminations of the arterial circulation, is the cause of the increased bulk of urine. (It differs not in character from the increased flow of water produced by ligation of the aorta below the renal branches; or from the increased tension produced by a draught of water, which augments the bulk of blood and causes greater tension on the glomeruli, and increased elimination of water, the amount of salts remaining entirely unaffected.) This increased tension tells on the arteries in time, and another morbid process is added to the rapidly widening circle of troubles. The blood, pumped into the elastic arteries by a hypertrophied left ventricle, while its efflux is opposed by hypertrophied muscular arterioles, unnaturally and unduly distends the elastic arteries. This leads to increased recoil, as long as their elasticity is unimpaired, and increased flow of blood into the coronary arteries and full blood supply to the heart itself. Thus the hypertrophy of the ventricle is maintained. But this increased distension of the arteries leads to parenchymatous inflammation in or under the brittle inner coat, or endarterium, and to the formation of that condition known as atheroma. The diseased arteries become elongated and widened from loss of their elasticity, and from imperfect recoil on the extreme distension. This affects the whole arterial system, gradually and slowly, but surely. During this time the increased arterial tension must itself be a source of danger, especially during exacerbations of the affection, that is, at times there is more imperfect depuration than usual, due to some disordered innervation in the kidney, probably, as we shall see in a little time, spasm in the hypertrophied arterioles, and obstructed blood flow, leading to violent efforts (palpitation)

in the hypertrophied heart to secure complete ventricular emptying in the face of the obstructed flow.

This tests the arterial system, and the weakest point may rupture; and that weakest point is to be found in the thin-walled arteries within the encephalon. Thus we get great liability to cerebral hæmorrhage, especially when the flow forward is obstructed by thickened arterioles in a state of spasm.

Cerebral hæmorrhage may occur from increased heart power alone, as in aortic insufficiency, without the effect of arteriole contraction, which adds most materially to the danger of rupture and true apoplexy. Another morbid action is now added to the list. Cerebral hæmorrhage is apt to occur at this stage, and is a great source of danger to life practically. It is more apt to occur in hypertrophy of the left ventricle, and is a direct consequence of that compensatory morbid action. For hypertrophy of the left ventricle is a directly compensatory and conservative action to meet obstructed blood flow. Where dilation results, cerebral hæmorrhage is certainly not so likely to happen, but then we get other and surer mischiefs in its stead. The imperfect circulation affects the system generally, for if the arterioles cut off the combustive changes, at one time a desirable action, they also cut off the blood supply at other times, when this impaired combustion is not called for. More especially too this dilated ventricle does not distend the aorta sufficiently to produce a recoil equal to overcoming the resistance offered by its own thickened arterioles. It is imperfectly nourished itself, and grows weaker, and so less perfectly nourished still, degeneration of structure follows, and death from heart failure takes the place of danger from cerebral hæmorrhage.

The changes in the two muscular extremes of the arterial circulation act variously, each at one time an advantage; at other times manifesting that they are pathological and not physiological processes. Thus, during an acute exacerbation or temporary increase in the usual blood impurity, the arterioles contract and reduce combustion, and thus allow the system to right itself of the accumulated products of histolysis; but

this repeated action leads to hypertrophy of their tunics, constant obstruction to the blood-flow, and to generally imperfect combustion, and thus further accumulations of imperfectly oxidised tissue-waste. For, as Bence Jones points out in his Physiological Essays, Bright's disease is a disease of suboxidation. So we see, that a process conservative at one time, is destructive at another. In like manner, when hypertrophy of the heart occurs it balances the circulation, and by increased power can drive an equal quantity of blood through a smaller tube in an equal time. Thus ordinarily the condition of normal health is closely approximated, but during the exacerbations, when arteriole spasm is met by violent ventricular contraction, the arteries are imperilled. This danger is obviated when dilatation is present, instead of hypertrophy, in the left ventricle, but, in place of danger during exacerbations, we find imperfect power to overcome arteriole action during the intervals, a general condition of suboxidation, further and more frequent accumulation of waste products, further arteriole opposition and greater dilatation following; and, consequently, not only a worse condition to meet and undergo any intercurrent disease, but positively a shortening of the time when heart failure will inaugurate the graver conditions, to be considered in the second section. Thus we see that the condition of hypertrophy in the left ventricle is a compensatory action suppositing lesser evils for greater ones, enabling the status of normal health to be more closely approximated, and certainly better enabling the system to undergo attacks of intercurrent disease than when dilatation is present. For when dilatation is present there is not only a less power to undergo intercurrent disease, but the general feeble condition and the accumulation of waste products in the blood, from suboxidation, render the system more liable to intercurrent disease, *i.e.*, it is induced by more trivial exciting causes.

The exacerbations of chronic Bright's disease are curious and interesting. As their pathology is not yet certain, the subject must be spoken of with diffidence and caution. We know, however, that during the long period of life over which this condition frequently extends, variations in the

urine are a marked feature. At one time the patient passes large quantities of pale-coloured urine of very low sp. gr., 1·003 or so; and at other times smaller bulks of urine than the normal with a very high sp. gr., indeed laden with waste products, urea, lithates, &c. Now, this is not a mere question of dilution, and that an equal quantity of solids give a low sp. gr. in large bulks of urine, or that the sp. gr. is heightened when the bulk of urine is lesser than the normal. There is a positive antagonism betwixt the amount of solids and the bulk of urine. Nor is it difficult to conceive this, for in the kidney we have seen there are two divisions of lesser arteries, one-half connected with the elimination of water, and the other with the nutrient processes and the elimination of urine salts. It is obvious, then, that any vaso-motor disturbance which would increase the circulation through the one set by dilatation of its walls, would decrease the circulation through the other set; the calibre of the renal artery remaining the same. There are variations of action in healthy kidneys which we, apparently very rightly, attribute to nerve action within the kidney itself; and these disturbances are naturally more marked in a kidney the subject of chronic disease, especially when its own arteriole muscular walls, the part affected by vaso-motor nerve influence, are themselves the subject of hypertrophy. Thus we can see without much difficulty thus far, viz., that these variations are likely to occur more commonly in chronic Bright's disease than in health, and that the variations are more marked too; and, further, we can understand that all the symptoms, resulting from the above-described pathological condition, are more aggravated during the period of time that the patient is passing large quantities of pale-coloured urine of low sp. gr., and that relief is experienced when small quantities of dense urine are passed.

Compensatory Actions.—When we remember the arguments of Carpenter in his article Secretion, in Todd's Cyclopedia of Anatomy and Physiology, viz., that each excretory organ is capable of supplementing another organ's function, and of eliminating materials special to another organ rather than itself, a property which they possess as mere modifications of

the general excretory surface in the lowest forms of life, we can easily understand, that in the condition of chronic Bright's disease compensatory actions in other organs, to supplement the defective action of the kidney, are very common. As we see the kidneys eliminate bile in cases of jaundice, so other organs eliminate urine salts in Bright's disease. Thus we see that not only are these sufferers more liable to intercurrent affections from ordinary causes, but that they are also liable to disorders of various organs, as uræmic diarrhœa, gouty bronchitis, gouty eczema, gouty arthritis (which Bence Jones says, truthfully enough, are compensatory peroxidations), which are not morbid processes *per se*, but compensatory actions. This is an important matter to bear in mind in the treatment of various affections in these subjects. Not only this too, but even affections arising as they do in sound persons and without any reference to the cachexia, are apt to assume a peculiar type, and persist as compensatory excretory actions; thus an ordinary bronchitis may assume a gouty type, and will only yield to remedies which affect the cachexia; and even an ulcer will assume this type and become "constitutional," as the late Professor Miller of Edinburgh was fond of terming it, becoming really an excretory organ, and its surface eliminating. The histological elements of skin are there, in another form merely, and we know from Funke, that in the elimination of urea, the phosphates, chlorides and sulphates of the alkalies, the constituents of sweat are those of urine (Dickenson). The condition of imperfect depuration of the blood thus leads to compensatory actions in other organs; and thus by reduced combustion, at times of great impurity, and these compensatory actions, the system preserves itself from such grave disturbance as must necessarily be fatal, though sometimes a condition may be reached, especially when a greater compensatory action than is common is required, when it seems almost impossible for the patient to survive.

Symptoms. In the Heart.—Thus we see in this combined condition of heart and kidney disease, that there are disturbances in various organs, resulting from the pathological changes, which may aid us in recognising this condition.

Thus we find that heart symptoms as palpitation, occasional irregularity, are present, and especially during the exacerbation; when the presence of the stimulus in excess leads to spasm in the hypertrophied tunics of the arterioles, increased obstruction to the blood stream, and imperfect ventricular systole and palpitation; the palpitation indicating the stress upon the ventricle from distension.

Thus palpitation is a common symptom in this condition.

Arterioles.—This spasm of the arterioles is often manifested by cold, "dead" hands or feet, so common in the sufferers from chronic Bright's disease. Probably this same condition in various tissues, muscles, tendons, or rather sheaths of tendons, nerves, &c., leads to the various painful affections, as rheumatism so-called, neuralgia, or sciatica, &c., and to these wasting of muscles from too excessive spasm, found in this condition.

Brain.—The brain is liable to suffer from the poisoned blood on which it must feed, in chronic kidney disease, and we find headache frontal, occipital, or vertical very common. Vertical headache is a symptom of very great importance in this condition, and is regarded as almost pathognomonic; indeed frequent and recurring vertical headache is a diagnostic symptom of the greatest moment. Cerebral anæmia is common, and may be partial, or so marked as to simulate apoplexy. It may only amount to a feeling of great prostration, nervousness, and palpitation, due to imperfect evolution of nerve force by the anæmic centres, as markedly seen in the cerebral anæmia, with melancholia, which accompanies poisoning with bile products, as well as the other cause of arteriole spasm.

At other times the condition of uræmic coma and convulsions may be produced; and it is yet an unsettled point whether these are due to the positive presence of the poison, or to the anæmia resulting therefrom, from arteriole spasm.

The mental manifestations will be considered further on.

Lungs.—There is a tendency to pneumonia from the poisoned blood condition, which may fairly be regarded as uræmic. But there are also curious "attacks of inexplicable dyspnœa" (Basham) occurring in this combined disease.

Niemeyer attributes this to œdema, of a transient nature, but though this is probable enough in the later stages of heart failure, it is improbable in the earlier stages. They are more probably connected with spasm of the pulmonary arterioles, as a form of apnœa.

Bronchi.—There is a liability to bronchitis, not only as a compensatory action, but as induced by this condition. Headlam Greenhow has shown the dependence of bronchitis on gout; and it is familiarly known that attacks of gout and bronchitis alternate. Ordinary bronchitis may take on the peculiar type, and be kept up by the constitutional condition. In many cases litmus paper will be reddened, and positively demonstrate beyond doubt that the mucus is no longer alkaline, but acid. Winter cough and expectoration are also very common here. In summer the skin acts freely, and the defective action of the kidney is supplemented so completely as to endow the sufferer with a very fair share of health and strength as long as the hot weather lasts; but as soon as it is colder the condition becomes worse, though eked out by attacks of what is called autumnal diarrhœa. In winter the supplementary action has settled on the bronchial lining, excited probably to some extent by the variations of temperature to which the bronchial lining membrane is exposed in winter. This is quite distinct from bronchial serous flow from venous congestion common in the later stages. A long fit of coughing, with expectoration, at rising in the morning in winter, getting rid of the accumulations through the night, is common here. We do not yet know the connection betwixt gout and true spasmodic asthma, or if any connection exists.

Stomach.—Derangements connected with the stomach are very common in chronic kidney disease; not the gastric catarrh of venous stagnation depending on heart failure, but a compensatory action. This is especially liable to show itself during the periods of digestion, when the stomach is normally acid, and is extremely intractable if the condition on which it depends be forgotten. At other times we find uræmic vomiting, the vomited matters being of a urinous character and smell, and Frerichs has claimed to have found

masses of carbonate of ammonia in the vomited matter. This uræmic vomiting is apt to occur in the gravest forms of uræmic poisoning, when the breath is also distinctly urinous, and is regarded as a serious condition.*

Intestinal Canal.—The readiness with which the mucous lining of the intestinal canal takes on compensatory action is well known. In practice, action on the bowels to elicit compensatory action is commonly resorted to in blood poisoning, and especially in uræmia. Thus in the uræmic condition which results from scarlatinal nephritis, purgatives by mouth, or clyster, are commonly resorted to. Spontaneous uræmic diarrhœa is not yet sufficiently recognised. This is more common than is supposed, and often accompanies acute congestion of the kidney. There is one difficulty in the recognition of it, and that is the diminished renal secretion. We are all so familiar with the fact that free purgation, with dilated blood-vessels in the intestinal canal, lessens arterial tension and renal flow, just as action of the skin does, that diminution of urine is apt to attract no attention as to its cause. This often leads to serious error in treatment, and fatal results; as pointed out by the writer in a paper on Uræmic Diarrhœa (read before the Medicine Section of the British Medical Association at Leeds, 1870).

Serous Membranes.—These are also liable to inflammation in the course of chronic kidney disease, as pleuritis, pericarditis, meningitis, peritonitis, and enteritis. Some persons seem to have a special liability to affections of their serous membranes in this state, and one relative of the writer's has the symptoms of most acute enteritis and peritonitis which seem as if about to be fatal. When profuse ammoniacal vomiting and purging come on, then relief is at once experienced, and improvement sets in, followed by a brief period of fair health, all things considered, until a feeling of malaise for some days precedes another eliminative explosion. Were it not that no attack has yet been fatal, the prognosis would

* My friend Dr. Charlton, of Newcastle, tells me he has found this condition only tractable by carbolic acid, which he regards as the best remedy. He even thinks if vomiting yield to carbolic acid, there is some ground for thinking it uræmic.

be unhesitatingly given of impending death, but the system seems to become habituated to these compensatory disturbances.

Joints.—The tendency for gout to settle in joints is well known, and equally well known is the fact that the most persistent form which retained products of histolysis assume is that of uric acid. The explanation given by Bence Jones seems the most satisfactory as yet. He thinks that uric acid is removed from the blood, when circulating in excess, by filtration into the tissues, especially chondroid; this proceeds to a point when inflammation is induced, and the increased temperature breaks up the uric acid into urea and water and CO_2; these soluble products dissolve, or are taken up by the blood and excreted; a cleansing process by peroxidation has removed the waste products, leaving no trace on the tissue, until repeated similar attacks have passed over them. Thus, he says, the inflamed fingers and toes are converted for the time being into supplementary kidneys. He regards the inflammatory affections as peroxidising depurative processes, occurring at intervals during the course of this chronic disease of suboxidation. The general arteriole spasm of this disease and its effect on combustion entitle it to be regarded as a disease of suboxidation, and the good effect of these peroxidising inflammations is apparent.

Hilton, in his work on Rest and Pain, has regarded the heart as structurally homologous to a joint, and that it is consequently affected by diseases which affect joints generally. Thus inflammatory attacks affect it in common with other joints in the course of acute rheumatism, gout, pyæmia, &c. Endocarditis, and especially over the valves, is, therefore, common in the course of chronic kidney disease, and adds valvular lesions to the heart troubles arising otherwise in this disease. It is more than probable that the force with which both aortic valves (by aortic recoil) and mitral valves (by increased ventricular systole) are closed when the blood stream is obstructed by arteriole hypertrophy, and intermittent spasm aggravating this action, is the cause of this valvulitis, so associated with strain.

Skin.—The effect on the skin of chronic kidney disease

varies. In some cases there is a decidedly dry, harsh state of skin produced by the obstruction offered by the thickened resistive arterioles, and here it is almost impossible to induce diaphoresis, and Professor Johnson has found that cold by inducing arteriole spasm, continued into palsy, and then dilatation, the most effective means of producing diaphoresis in these cases. At other times the skin, from its depurative action being so closely allied to that of the kidney (Funke), is liable to suffer from eruptions, and especially eczema, which are eliminative in action. It may be made a question how far the skin in carrying out its function becomes inflamed and eruptions form, from the imperfectly depurated blood with its excess of urine salts acting as an irritant; or that it is, from being fed on impure blood, more liable to inflammatory attacks; but certain it is that the eruptions are maintained by the excreta which find here a channel. At other times acute dermatitis, commonly called erysipelas (the non-specific form), ensues, which may end in gangrene (Rosenstein), or the formation of ulcers. Carbuncles are commonly found in gouty subjects, or these with Bright's disease. Griessinger has thought purpura hæmorrhagica to be connected with chronic kidney disease.

The Hair.—As a mere modification of epidermis the hair is affected in chronic kidney disease. Laycock says, that when the hair is fine and soft it is apt to fall off, especially from the vertex, and form a mere fringe round the skull, like a monk's tonsure; where strong and thick it remains full and strong, but loses its colour and turns rapidly white. It is impossible to pay any attention to physiomonical diagnosis without being convinced of the correctness of Professor Laycock's observations. In addition, I may venture to add that intensely bright white hairs in a head covered with profuse raven locks are very closely associated with chronically diseased kidneys, an impression much strengthened by observations made in the Pathological Institute of Vienna.

The Ear-lobe.—Professor Laycock has laid stress on the diagnostic value of a full, firm, red, rotund ear-lobe as an indication of gout, and certainly it is a characteristic of gouty families, and found in gouty people, and it is not easy

to separate gout from chronic Bright's disease (Garrod). In some small, dried-looking people with this renal disease the ear-lobe looks withered and shrunken, as if it suffered from something allied to cirrhosis.

There are groups of pathological changes which indicate, even to the eye, the existence of chronic Bright's disease. In some there is a florid complexion, a full pulse with left ventricle hypertrophy, atheromatous, dilated, capillaries on the cheek, full alæ nasi and ear-lobes, bald scalp, with a fringe of grey hair extending from the temples round the occiput, full temporal artery, a plethoric apoplectic constitution, with sanguine temperament, and hasty irascible temper. Along with this are found uric acid sediments in the urine, and the patient invariably gets up at night to make water. This last symptom is curiously associated with Bright's disease, and, when freed from sources of error as diabetes, imbibition of large quantities of fluid at night, or later evening, may fairly be regarded as pathognomonic.

In others there is a different grouping of symptoms. The patient is thin, not at all plethoric, nervous, excitable, with a bushy head of grey or white hair, and with small, withered ear-lobes; apt to have gouty dyspepsia, jaundice, or skin affections, and, if advanced of life, a well-marked fatty arcus senilis. The patient has palpitation usually. Getting up at night to make water is also found here. The general appearance of the patient is that of a withered apple, and gives the impression of a general development of connective-tissue which has shrunken and given this dried-up, wrinkled appearance to the tegument and external parts generally.

At other times the pallor of skin, so often remarked in Bright's disease, is marked; there is coldness of extremities, and often "dead" hands and feet, chronic bronchitis, cough, heart failure, dyspnœa easily induced, and the subcutaneous areolar tissue fatty, and œdematous looking; there is fulness beneath the lower eyelids, and a watery looking state of the tissues of the face, which often will become inflamed by exposure to a sharp cold wind. These patients, usually females, are the people so often met with in the upper and middle classes, who catch cold on the least exposure, have facial

erysipelas from facing a wind coming from church, are liable to recurrent diarrhœa, or skin eruptions, are irritable, sleepless, and "whimmy." Here there is also nocturnal flow of urine so marked as to attract the patient's attention.

Of course the sufferers from chronic Bright's disease do not constitute three groups, or any other number of groups, these are merely illustrations of people who have come commonly under the writer's notice as subjects of this affection.

The Eye.—There are changes in the eye connected with chronic renal disease, as rapidly increasing impairment of vision, want of accommodation in the eyes, and seeing double, each eye having its own impression. The changes to be observed by the ophthalmoscope are described as usually white stellate spots on the retina, with a swollen-looking condition of the retinal vessels. Rosenstein describes the eye affection as retinitis, and it is also called retinitis apoplectica in consequence of the tendency to the formation of apoplectic dots, especially when the left ventricle is distinctly hypertrophied. The white spots are generally attributed to formation of connective tissue, which is undergoing, or undergone, fatty degeneration. The subject is considered at length in works on Ophthalmoscopy and Diseases of the Eye, and will be found in Clifford Allbutt's book, or recent editions of Jäger. A form of uræmic cataract, from infiltration of urea, allied to diabetic cataract, is also found. The appearances in the eye are often such as to, alone, justify the ophthalmoscopic observer in pronouncing positively as to the existence of renal disease.

Kidneys.—The effect of the progress of this affection is to influence the renal secretion. This is increased in volume, especially in the middle stages of Bright's disease, so markedly increased in volume as to be characteristic, and is a symptom of value in diagnosis. There are also remarkable variations in the bulk and sp. gr. of the urine, at times the bulk is extreme, and the sp. gr. very low indeed; at other times the bulk of urine is abnormally small, and the amount of urine salts disproportionately large. It would seem that there are more marked results of nerve-disturbance within

the kidney itself in this condition of hypertrophy of the muscular walls of the arterioles than there are in health. That nerve disturbance, how induced cannot yet be detected, causes these variations is almost beyond doubt. For though increased tension occurs and would produce increased pressure on the glomeruli, if the renal arterioles did not share in the general vaso-motor spasm, and consequent hypertrophy, still when the renal arteriole walls are also in action, this increased tension is met by diminished calibre in the vessels leading to the glomeruli; we cannot then attribute the increased bulk of urine to mere increased tension in the paroxysms. There is evidently at that time spasm of the nutrient branches, and thus the calibre of the renal artery remaining the same, increased blood circulation through the convolute branches. This leads to imperfect elimination of urine salts by the distribution of the nutrient arteries, blood impurity increases, general tension is increased, and the pressure on the glomeruli follows the greater arterial tension, and there is a still larger bulk of urine passed. The attack passes off by diminished bulk of urine and large amount of renal salts. The spasm in the nutrient arteries gives way in time to dilatation, and then the position is reversed, the nutrient arteries and arterioles are dilated, and the calibre of the renal artery remaining the same, there is increased flow through the nutrient vessels with large elimination of urine solids, while the circulation in the convolute vessels is lessened and the bulk of urine diminished.

The nerve-disturbance within the kidney is the commencement of the paroxysm, and the consequent more imperfect depuration of blood keeps it up.

And now as to the increased bulk of urine normally, if the expression may be permitted, existing in developed Bright's disease. We have seen that the calibre of the renal artery remaining the same, but its peripheral distribution being diminished by chronic destruction within the kidney, the pressure would be increased in what remained sound, and the bulk of urine be the same. But it is increased! It is evident then that there must be increased arterial tension, and thus increased pressure on the renal artery and its dis-

tribution. This would be accounted for by the hypertrophied left ventricle so commonly found. But more observations are necessary, and some very exact ones are desirable as to the existence of this increased bulk of urine, along with hypertrophied left ventricle, and as to what effect is exercised by a dilated left ventricle. The observations and experiments of Phaff, Winogradaff, Goll, Ludwig, and Traube, are demonstrating the connection of water elimination by the kidney with arterial tension, almost beyond question; and the effect of cardiac dilatation on the bulk of urine in chronic renal disease ought, therefore, to be conspicuous. If found to diminish the bulk, while hypertrophy increases it, it would go far to remove the excessive water elimination from the kidney itself, and locate it in the heart, which is secondarily affected.

That the bulk of urine should ultimately in the later stages be diminished, it is not difficult to comprehend, when we remember that the heart hypertrophy fails when the arteries become extensively diseased, and that then there is venous congestion, which diminishes arterial tension, for the fuller the veins, the emptier the arteries, while the auxiliary forces of more extensive kidney disease, and the massive hypertrophy of the kidney arterioles themselves (Johnson says the kidney arteriole muscular hypertrophy is often more marked than is that condition elsewhere), must aid in producing diminished water elimination; these combined forces much reducing the pressure on the glomeruli.

We can also understand that suppression of urine, the ischuria renalis of Mason Good, may result from spasm of the hypertrophied tunics of the convolute kidney arterioles. Indeed it is difficult to understand how so purely physical an action as water filtration through the glomeruli of the kidney could be arrested, except by completely cutting off the blood from the glomeruli. In this case we must see that there would be rapid circulation through the vasa recta, or nutrient branches, else there would such congestion of the kidney ensue as must necessitate complete disorganisation; consequently there must be free blood supply to the nutrient branch-distribution and to the epithelial lining of the tubuli

uriniferi; but without water to wash out the salts none can be removed. This condition with its subsequent blood-poisoning, uræmia, diarrhœa, vomiting, and other compensatory actions, is not unfrequent in the course of chronic Bright's disease.

But as to increased flow of urine at night and the patient having to get up to make water, it is inexplicable by any action within the kidney, or question of increased pressure on the kidneys from their more dependent position below the aortic blood column, raised on the spinal vertebræ, in the recumbent posture. Is it an actual increase at night? or that the urine flow is more noticed, from the trouble of getting out of bed which it entails? This question must be decided ere we can seriously seek for the explanation of a condition which is not as yet an ascertained fact. That the bladder is more intolerant of a diluted urine than a dense one, we have the word of a very competent authority, Sir Henry Thomson; we can understand a bladder more rapidly filled, owing to the general water elimination here being increased, with a fluid of which it is less tolerant, the water being of low sp. gr. of this condition, making its solicitations more urgent, and necessitating attention to its calls. But do the sufferers equally get up to make water during the time of exacerbation with large bulk of dilute urine, and also in the opposite condition of small bulk with great density? That getting up at night to make water is one of the most common and trustworthy symptoms of chronic Bright's disease is well known, let its explanation be what it may.

There is also often seen an increase in lithates and in uric acid itself in this disease. And whether urea and uric acid are formed from the earlier products of histolysis, as creatine, creatinine, &c., by the kidney itself, as the experiments of Zalesky and others would tend to show; or are eliminated from the blood by the kidneys merely, as other experiments would seem to assert; it is equally certain that extensive kidney disease impairs the elimination ordinarily; and during the periods of dilatation of the nutrient arteries and dense urine, lithates and uric acid itself are commonly found.

Uric acid is a result of imperfect oxidation we are generally taught, though Prout thought it the histolytic termination of gelatinous textures, and consequently it is here, free, or in combination with some base, soda, potash, ammonia, or lime (Rosenstein).

A shrewd north country doctor, the late Dr. Pearson, of Penrith, gave the prognostic value of these sediments very shortly: "It is all right when you do see them, but the trouble is when you don't see them;" and it is not easy to improve on this. Calculi in the tubuli uriniferi are found sometimes, usually black, and sometimes in cystic kidneys, as large as peas, or they drop into the pelvis of the kidney, and grow there, or pass down the ureter, often giving no little pain in doing so, and either lodge in the bladder and increase in size, or pass out by the urethra. Uric acid forms large crystals out of solutions, and these are found in the urine, after it has stood and cooled, large enough to be recognised by the naked eye; much resembling coarse Cayenne pepper grains.

Sometimes the imperfect oxidation results in the formation of oxalates, now regarded as a mere modification of urates, with an identical significance.

Tubé Casts.—These are the most convincing evidences of chronic Bright's disease when found. But even to this evidence there are drawbacks; firstly, they are very difficult to find very often, being in much smaller numbers than in catarrhal, otherwise called tubular, or desquamative nephritis; and, secondly, when found can give no information, as to the amount of kidney diseased, or as to whether the disease is common to both kidneys or confined to one.

These are serious drawbacks, and leave us in great doubt as to the amount of disease present, which must be gathered or surmised rather from the general symptoms. The casts are small and granular, apt to be broken from their brittleness, either in the uriniferous tubules, or elsewhere, including under the glass cover of the microscopic slide, and are then found in small masses. Their appearance and the changes in their appearance, are well given in Beale's book. When new cylinders are found, the exudation casts, or fibrin-cylin-

ders, they testify that some new portion of kidney is being implicated, and along with them will be found albumen. During the later stages, to be considered in Part II, these exudation casts are common, resulting from venous stagnation.

Albumen.—The first discovery of albumen in urine was hailed as an infallible test for kidney disease, whose utterances were unmistakable. The presence or absence of albumen in urine are not now so confidently regarded as aids to diagnosis. It is a well-known fact that in chronic parenchymatous, diffuse, or interstitial nephritis, as it is variously named. albumen may be absent for long periods; and that when it does occur, it is usually slight in amount. Without entering into the difficult question of the diagnostic value of albumen in the urine, it may be desirable to briefly consider it here as far as it bears on the subject under discussion. In the first place, its absence for long periods proves most conclusively that it is not an accompaniment of a structurally altered kidney, where one or more localised tracts are destroyed and encapsuled by connective tissue; this is no more than we could conjecture from reasoning on the matter. This portion is utterly cut off from all possible function, and can, therefore, have no symptom derived from disturbance either in its function, or in what it was once functionally connected with. While as to increased arterial tension Niemeyer and Rosenstein alike assert that no albumen has ever appeared, in the urine, in all the experiments of ligaturing the aorta below its renal branches. Increased arterial tension does not, then, produce albuminuria. There is only one conclusion on the matter, and it is this, albuminuria, in chronic Bright's disease, indicates the implication of a hitherto unaffected piece of kidney: it demonstrates that another tract is being added to the portion previously destroyed. Thus it is usually found in small quantities and with a few exudation casts, or fibrin-cylinders. These signs indicate an extension of the disease, and therefore are of evil prognostic, as well as diagnostic import. Albumen may indicate a more general implication of the structure of the kidney at other times, and is constantly present in the later

stages of venous congestion. But in the diagnosis of chronic Bright's disease, while the heart is strong and equal to its labour, we derive no aid from albumen in the urine; its absence can give us no comfort, nor does its presence tell us how much previously sound kidney is being involved. During the ordinary progress of the case albumen is ever absent from the urine.

The varying amount and quality of urine, its increase in bulk, and its call at night, the occasional suppression, complete or incomplete, must often be the sole indications derived from the kidneys themselves; excess of lithates or oxalates, at times, and albumen and tube-casts (exudation) aiding us at intervals. Old granulation casts are found with too much difficulty to be commonly at hand, but speak positively to the existence, but not to the extent of disease.

Dropsy.—This is not a characteristic of chronic kidney disease so much as acute attacks of nephritis. It is rather connected with the face than the lower extremities, which are the earliest seat of œdema from heart failure. It is more marked in the face in large white kidney than in small granular kidneys. In the latter it is only usually found as a slight puffiness beneath the eyes; while in some cases it seems to exist as a watery state of tegument, and with serous flow, readily induced. At other times it is more general, even when there is good action of the heart and firm incompressible pulse. It is not here to be confounded with the more truly cardiac dropsy of the later stages, which is merely modified by old kidney disease. Long arguments have been entered into as to how dropsy thus occurs, but no satisfactory emergence has yet been achieved. It has been supposed that the amount of urea in solution causes the water to readily leave the vessels for the areolar tissues; dropsy is certainly at times a sort of compensatory excretory action. Bristowe (Croonian Lectures, British Medical Journal, April 27, 1872) says of retained, or rather non-eliminated water, in chronic kidney disease, "a great deal no doubt passes into the connective tissue, and accumulates there, the capillary system throughout the body acting as a kind of general excretory organ, and hence results general dropsy;" and with the expression of the fact

we must, at present, rest satisfied. That dropsy occurs even with good heart power and free evacuation of water is one of the circumstances about it which is most inexplicable; and altogether the subject is not an easy one. The younger reader, who wishes to know more about dropsy and its relations, can profitably and pleasantly spend some spare time in perusing and conning the pages of a very excellent book by Basham, "On Dropsy, and its Connection with Diseases of the Kidneys, Heart, Lungs, and Liver."

Mental Manifestations.—When first beginning to study chronic kidney disease, and especially in reference to heart disease, the writer was struck with the occasional irritability, active and positive, manifested in the subjects of this diseased condition. It was not the imperfect thought of the anæmia of heart failure, which is rather a condition approaching amentia, and depends on lack of arterial blood, but a condition of mental activity presenting some peculiar features. Irritability, disorder of temper, amounting to positive unreasonableness, great annoyance at small matters, which "put them out" to an extent quite disproportioned to the slight exciting cause, characterise this affection and especially its exacerbations. A dear relative of the writer's used to describe it pithily by saying that "she could fight with a feather." The positive mental suffering and annoyance are aggravated by a consciousness that there is unreasonableness in it, and that it is a something morbid. This condition completely baffled all speculation until a perusal of Bence Jones's Physiological Essays, where the connection of this irritable mental condition with oxaluria and retained lithic acid was described. There is no doubt this mental irritability arises from the brain cells being fed by impure blood, and that the retained products of histolysis are the foundation of it. Since then more accurate and extended observations enable me to speak positively to this association, and to connect periods of unusual irritability with paroxysms of exacerbation, when the patient passes large quantities of pale urine of very low specific gravity; much alleviation being experienced when the spasm yields, and a discharge of urine of small bulk, but intense saturation fol-

lows. The effect is to disturb the mental equilibrium, and has different results, sometimes not altogether unfortunate, but most decidedly to be regretted.

It seems possible to charge some mental actions, otherwise inexplicable and unaccountable, to retained urine salts, and especially to uric acid, a conclusion perhaps more acceptable to charity than likely to be accepted by psychologists. It would often be satisfactory and agreeable to explain anomalous and indefensible acts by this theory, and lay some of human frailty to the charge of uric acid.*

Before proceeding to the diagnosis and prognosis of this combined condition, it may be instructive to some readers to sketch an outline of some cases as they present themselves in practice. No completeness is aspired to, either in detail or variety of groupings of symptoms, but just enough to aid some younger readers to understand some cases which may, without these hints, give them much trouble, often futile. This has the advantage over long clinical cases of occupying less space, if not quite so satisfactory. These are mere outline reminiscences of cases which gave great trouble till the clue to them was obtained.

Nervous.—A person of good nervous development, or rather of the nervous diathesis of Laycock, when suffering from an exacerbation presented the following characteristics:—Vertical headache, great and unusual mental irritability, marked sleeplessness, not due to the headache,

* It may seem somewhat out of place to allude to impressions formed by the writer as to the effect of retained urine salts on mental processes here, even in a foot note; but this excess of urine salts does seem to have a stimulant effect on the brain, and gouty people are usually possessed of some talent. The conclusions, so far, seem to indicate that many persons of good brains, but lacking in energy and inclination to think, are stimulated by retained uric acid into excellent thinking, and attain a reputation late in life. While in others, with small irritable "foxy" brains, the disturbing effect of these retained excreta makes the cares of business, &c., quite intolerable. Retirement from business at first gives relief; but soon this irritability incites them to have something to do, and this too commonly is effected by becoming members of boards and committees, when this mental irritability takes the form of mischievous perversity, of ill-controlled interference with everything and everybody. In this condition they remind the writer of nothing so much as a cancerous gland—no longer fulfilling any useful purpose, but merely a source of irritation to everything around them.

which was not pronounced, great flow of pale urine of low specific gravity, palpitation, loss of appetite, and general uneasiness. Usual state: active habits of mind and body, with a dilated heart with some hypertrophy. This peculiar grouping of nervous symptoms recurred again and again.

Sleeplessness.—This is a marked characteristic of chronic Bright's disease. It is not connected with pain or bodily discomfort, but is provokingly persistent. It seems due to the irritation exercised by impure blood, and is aggravated by thinking; which is ever unsatisfactory, inconclusive, and tending to work round and round in a circle, the same point being reached over and over again, but no conclusion ever attained. This condition becomes even more marked when heart failure entails imperfect supply of arterial blood as well as a spanæmic condition. For the blood tends to positive thinning, anæmia, as well as containing excreta in excess.

Diarrhœal.—Here was a good physique, with high nervous development. There was marked hypertrophy, arterial atheroma, large elimination of pale urine, great mental irritability, aggravated in a paroxysm, and during the paroxysm a nervous characteristic cough, excitement, and supplementary catharsis. The diarrhœa for long protected the patient, who suffered from one attack of cerebral hæmorrhage, until a general peroxidation or inflammatory eliminative action, commencing with erysipelatous inflammation of the integument, with sloughing; and ending in a typhoid condition, with incomplete suppression of urine, and uræmic diarrhœa, which terminated fatally.

In others dyspepsia will be combined with the general symptoms of Bright's disease, the peculiarities in the renal secretion, irritability, often with atheromatous capillaries in the cheeks: this chronic dyspepsia during the paroxysms becoming aggravated, with acid eructations. This pyrosis or water-brash is regarded by Rosenstein as very common in diffuse nephritis.

Bronchitis, generally sub-acute, and at other times acute, usually most marked in winter, will be found in other cases. It is aggravated by sudden falls in temperature, and arrested action of the skin. The accompanying symptoms or its

interchange with attacks of gout, a well-known interchange, will usually indicate its nature. It is quite unconnected with the serous flow from the bronchial veins in the venous congestion that follows the heart failure which inaugurates the second stage, according to the division here adopted.

This susceptibility to changes of temperature in the subjects of chronic kidney disease is decided and unmistakable. Dickenson in his work, "Albuminuria," alludes to this, and states its more marked character than the similar condition of chronic rheumatism. In the subjects of Bright's disease, sudden changes of temperature, by arresting the compensatory action of the skin, will often produce the most serious consequences; and a sudden fall of temperature will often furnish simultaneously a group of patients so troubled, each with their characteristic compensatory action, or so-called recurrent ailment.

In other cases there is found a condition simulating muscular rheumatism, often accompanying arthritis of the hip joint. There is general muscular pain and stiffness, or perhaps a group of muscles only are affected, with, sometimes, wasting from prolonged arteriole spasm, inactive skin, free flow of pale urine, great susceptibility to cold; and very frequently those curious pains which herald changes in the weather, and are probably connected with the magnetic perturbations which are the usual precursors of atmospheric changes.

At other times a patient will present these combinations of peculiar renal flow, especially at night; sleeplessness, physiogmonical indications, with heart hypertrophy and aortic atheroma, who is subject to recurrent inflammatory attacks, pleuritis, pneumonia, enteritis, &c. Here the concomitants will indicate its connection with the cachexia of chronic Bright's disease.

One singular manifestation is liable to occur in the course of this chronic disease, and though it is not quite in place to consider the various views of uræmia here, it is scarcely possible to avoid it in order to put the question fairly before the reader. Uræmia and uræmic coma are discussed by many English writers, and Roberts, in his work on Diseases

of the Kidney, gives a good *résumé* of the views held by various writers; while the philosophic essay of B. W. Richardson, in his asclepiad, may be studied with advantage, and other recent articles are of value. The exposition of Rosenstein as to the various views held, will be followed here with some additions to its variations.

Theory of Uræmia.—When the chemical results of mechanical changes, to use the phraseology of Bence Jones, first were studied in reference to the peculiar cerebral attacks found to frequently result from chronic renal disease, it was conjectured by Christison, that blood-poisoning by urea in excess, was the cause of the brain symptoms. Owen Rees thought it due to hydræmia, from thinning of the blood; and Osborne regarded the cause to lie in the membranes of the brain being affected, as by arachnitis, &c. The view of Christison found most favour. Some time afterwards Frerichs enunciated his well-known views of the decomposition of urea into carbonate of ammonia, being the cause of the symptoms. This view has circulated most extensively, but though it has been supported by Vogel, Petroff, and others, Hammond, in America, and B. W. Richardson, in England, have effectually demonstrated the unsoundness of Frerichs' view; while Zalesky followed Petroff's experiments without arriving at his conclusions. Injections of urine into the blood are supposed to insure uræmic attacks more certainly than injections of urea (Rutherford Haldane), and Vauquelin and Segalas tried unfiltered urine, and produced death by mechanical plugging of the capillaries of the lung. Filtered urine in the hands of Courten, Gaspard, and Frerichs, produced no head symptoms.

Further experiments were performed by Meissner and Voit, Oppolzer, Hoppe, Stannius, Schottin, Scheven, Perls, and Zalesky, with the result of finding these head symptoms follow nephrotomy or ligature of the ureters. From Zalesky's experiments, it would seem that nephrotomy or ligature of the renal artery were very quickly fatal, and the earlier products of histolysis, creatine, creatinine, &c., were found in the muscles and blood; while on ligature of the ureters the kidneys still seemed to exercise some function in converting

these earlier products into the more oxidised ones, urea and uric acid; which seemed to affect the result by being less rapidly fatal than the earlier products, when in excess. From this we may conclude pretty certainly that the earlier products of histolysis are more active than the more advanced ones. In addition to these views of a poison in the blood, the hypothesis of anæmia from hydræmia and loss of albumen has been started. Then the theory of anæmia from effusion into the brain substance (Gehirnœdem) has been evolved by Traube, who has found this cerebral œdema ever present in fatal uræmia. His view is that there is effusion into the brain substance, and being enclosed within the bony cranium, not allowing of expansion, the result was compression of the blood-vessels and cerebral anæmia. Rosenstein's conclusion is that Frerichs' carbonate of ammonia hypotheses must be abandoned; and that the hypothesis of the head symptoms being due to urea or other urine products, are doubtful and improbable; and that cerebral anæmia from brain œdema, or otherwise, is most probable.

That in fatal uræmic coma brain œdema is present, according to Traube's theory, is admitted in La Charité, Berlin; but as to whether the brain œdema causes the anæmia, or spasm and anæmia lead to œdema into the nerve structures within the skull, could be argued as long as either side pleases; and the argument of the cranium resembling an inverted bell-jar, and consequently that atmospheric pressure ensures its contents being ever the same in bulk, might be appealed to by either side. If the spasm of the arterioles lessens the bulk of arterial blood, some compensatory fulness must follow, and *vice versâ*, brain œdema must compress the blood-vessels and produce anæmia. The conclusions which seem to the writer most probable are these: 1st. That the anæmia is due to arterial spasm; 2nd. That this anæmia causes the symptoms of convulsions and coma; and 3rd. That this leads to œdema in fatal cases.

For the truth of this hypothesis may be adduced the effect of retained excreta on the vaso-motor system, and arteriole contraction, which is left out in Traube's view of the brain œdema producing the anæmia.

Then in a typical case of uræmia, a well marked case, which was the subject of a clinical lecture by Duchek, in Vienna, the loss of consciousness and tremors, incipient convulsions, were momentary and transient, like the lifting and falling again of a cloud, and more in unsion with the idea of intermittent spasm, or variations of completeness in spasms, than the constant pressure of œdema. In this condition recovery is possible, even when the spasm persists without remission or intermission; but œdema, effusion into the brain substance, is a result which ensures death. In the cases which do not terminate fatally, the diminution of arterial bulk by arterial spasm is probably accompanied by venous congestion, a condition not incompatible with recovery; when œdema sets in and produces persistent pressure on the blood-vessels, death follows.

This discussion has taken up more space than was anticipated, and the fact that uræmia, as seen in the more acute conditions of uræmic coma and convulsions, and in typhoid symptoms, is rather connected with urea, or even the earlier products of retrograde tissue-metamorphosis, creatine, and creatinine, while the more persistent tissue changes and chronic inflammations, of joints, &c., arise from uric acid, must stand unsupported, and be left to its intrinsic truthfulness. These acute conditions are associated with the accumulations of the more active earlier products of histolysis, while the more chronic parenchymatous inflammations, &c., result from the most persistant form assumed by this tissue-waste, namely uric acid.

Diagnosis.—This is not an easy matter, especially for beginners, who find it difficult to find the exact value of y (the diagnosis) from an indefinite number of x s (the symptoms) of undetermined value.

The symptoms and signs which go to form the diagnosis are numerous and of varying value, and their grouping and association will often have to settle the question; more or fewer being found together, and then there is often a suggestive relationship, which creates a fair presumption, casts, ophthalmoscopic signs; feeling the altered bulk of the

kidneys being alone positive, and this is often impracticable.

We will proceed with a brief *résumé* of the signs first. In the diagnosis of diffuse nephritis Rosenstein places the heart changes first. There is alteration in the left ventricle, usually hypertrophy, with decidedly accentuated aortic second sound, without apparent cause, and commonly increased impulse, &c. The pulse is hard, the radial artery rigid, and the arteries atheromatous, the temporal often being conspicuous, tortuous, and thickened. There is often a dry harsh skin, with atheromatous capillaries on the cheek, and "dead" hands and feet. The hair and ear lobe often aid. At other times a thin skin, with fulness under the lower eyelids, and apparently œdema in the subcutaneous areolar tissue is found. These are the changes in the eye, very positive when present. Variations in the bulk of urine, with antagonism betwixt bulk and specific gravity, and the curious symptom of getting up at night to make water, ever suggestive, and associated with the general increase in the renal flow. Uric acid crystals, &c., in the urine are of service in determining the diagnosis at times, and casts settle the existence of the disease, that is, old granular casts; new exudation-casts and albumen mark new implication of previously sound kidney. In addition to these, in thin persons it is possible to test the condition and bulk of the kidneys by touch, and play the kidneys betwixt the hands and fingers (Sir William Jenner's Lectures in Lancet, 1865). More or fewer of these are present, and along with them some of the following symptoms:—Palpitation, especially during exacerbations; vertical headache, very important; sleeplessness; mental irritability; and occasional curious attacks of dyspnœa. Disturbances of vision, as quickly increasing loss of vision, not being a visible affection of the eye, double vision, occurring in persons over middle age, are very suggestive. Then there come the effect of variations of temperatures, and muscular or other pains, recurrent attacks of gout, bronchitis, dyspepsia, diarrhœa, vomiting, other distinctly inflammatory affections, ever suspicious; tendency to anœmia in some cases, and

lastly, more rarely, attacks of uræmia affecting the cerebro-spinal nervous system.

Sex.—Perhaps of the two sexes men are more subject to Bright's disease; but sex is absolutely valueless as a diagnostic indication.

Age.—Advancing age ever gives a predominance of degenerative and chronic diseases; it is not only more liable to them, but survivorship entails even changes as time rolls on. Young people rarely suffer from chronic kidney disease, unless preceeded by an acute attack, and the same holds good of heart affections. Still both are found, not combined, true, as diseases from which even intrauterine life is not secured. The most common subjects are in middle life and more advanced age. Of course in making any such statement the fact is not lost sight of that the earlier and less pronounced stages are usually overlooked, and that, too, not from negligence on the physician's part, but in consequence of the insidious nature of the complaint itself. When this condition first commences is, in the majority of cases, not only unknown, but positively undiscoverable. We only detect it usually by the oncome of the various changes which follow in the wake of chronic kidney disease, or by the accidental discovery of a characteristic tube-cast. Indeed, the diagnosis is itself a surmise of greater or less probability; much depending on the skill, experience, and attention paid to the subject on the part of the medical man himself. There is a something in diagnostic skill not to be communicated to another, and, as Carlyle says, the eye can only see what it has learned to see. The effect of individual experience, and diagnostic as well as therapeutic ripeness in skill, are not to be acquired otherwise than by the sweat of each individual brow.

Prognosis.—There is no more difficult matter, in this combined condition of heart and kidney disease, than arriving at a fairly correct prognosis. The different factors are to be gathered from the elements constituting the diagnosis, the condition of the heart, its maintenance of its own structural integrity, the presence or absence of dilatation; the character of the renal flow in bulk and specific gravity, the probable amount

of disturbance of balance betwixt the amount of tissue-waste and the means to eliminate it, as gathered from the different compensatory over-actions of organs, or eliminative peroxidising inflammations, &c. There is no certainty, however, that the destructive process is not more steadily going on in those systems which are not disturbed by these intercurrent affections, than where the system is often so disturbed. The effect of these intercurrent attacks is to produce a cleansing effect upon the blood, and they are thus useful; while other systems may be preserving a fair amount of bodily vigour, and often great intellectual activity, as long as the supply of arterial blood to the brain is good and fairly maintained; but in them a steadily downward progress of increasing degeneration of one organ may be telling on another, and a general unobserved molecular decay produced, which will manifest itself in sudden failure of heart or arteries; or in some intercurrent attack rapidly becoming fatal, and revealing the unsuspected impairment. Indeed the prognosis must largely depend on the peculiar circumstances of each individual case, and to some extent on the skill of the practitioner in awarding to each symptom its true appropriate value. There are still elements of error, which elude all calculation and foresight, and the uttered prognosis must, in most cases, contain a saving clause.

Intercurrent affections arise commonly in this condition, and that is easily conceivable. For not only is the imperfectly depurated blood itself a source of irritation to various tissues which put on uræmic inflammation, but also tissues fed on this impure blood possess less resisting power to withstand ordinary exciting causes of disease. The system is more easily thrown off its balance, and the margin within which the disturbed balance may safely rock, without exciting disease, is distinctly diminished. Intercurrent affections are more dangerous to life than when occurring to a perfectly healthy person, in the first place; and are also more likely to persist in a chronic form, from taking on peculiarity of action and becoming eliminating surfaces, and thus tend to assume confirmed structural changes. The prognosis of an intercurrent affection is worse than when the system is free from

these consecutive morbid changes, though many of them are compensatory and conservative.

A totally different prognosis exists, however, in those intercurrent diseases, which may fairly be laid to the account of the waste-laden, imperfectly depurated blood. Here the system, though no doubt affected by the acute disease, is really being relieved from an accumulation of waste, and the inflammation is a depurative process. Many very grave and serious illnesses in persons of middle age and advanced life are to be thus regarded. There is, too, in these cases a tendency to recurrence, a point of some prognostic importance, as well as diagnostically valuable. In such cases the compensatory over-action, or inflammatory depurative process, will often bring the patient to death's door, and leave him there; just as all hope must apparently be abandoned, an improvement sets in, and gradually goes on to recovery. It is like a disturbed balance, the most dependant point is immediately followed by an upward rise. To a stranger called in these cases are very difficult to estimate, and no one, stranger or not, can tell at which one of these attacks the balance will be permanently overthrown; and the point of most depression no longer followed by an upward movement. Repetition of the attacks must be allowed to influence the prognosis, and the medical man who becomes familiar with these attacks in his patient, is not so entirely devoid of hope as a person not so familiar with them would justly feel. Thus the old medical attendant in such cases from experience of former attacks not being fatal, however alarming, gives a hopeful prognosis, which, being verified by the result, gets him the credit of "knowing the patient's constitution;" and not altogether without deserving it. For though, perhaps, he has had something else to do than meditate on morbid actions and reactions, on compensatory actions and conservative processes, as distinguished from morbid processes, and is unacquainted with the most recent views on peroxidations; he has learnt that in this particular case the grave, and, apparently, almost necessarily fatal attacks seem to exercise some peculiar effect over the patient, who not only recovers from them, but seems positively to have a

period of unusual good health for some time after each attack. The recurrence of them seems to confer on the system a sort of protection, from habitude, and these grave disturbances would, in all probability, be quickly fatal in a system not previously habituated to them. Mistakes are often most instructive, and the recital of the following one may possibly be of some service to younger readers, as it certainly has been to the writer:—Some years ago now, the writer was called in to attend an old lady of 72, who had pallor and swelling beneath the lower eyelids, night-voidings of urine, great variations in its amount, and a dilated heart, with a very irritable temper. There was also a history of preceding attacks of nephria and albuminuria. She was suffering from almost total suppression of urine (and the small amount, which could be secured, was slightly albuminous, and contained stray granular casts), and a profuse exhausting diarrhœa. Having some short time before been much taken up with uræmic diarrhœa, the writer declined to arrest the diarrhœa until the flow of urine was re-established. Acetate of potash, spirits of juniper, and buchu were given, the skin was acted on by hot bottles in bed (Sir James Simpson's bath), hot poultices faced with mustard were laid over the loins night and morning, and poultices without the mustard continued during the intervals; gin and water, milk, and beef-tea constituted the sustenance. For two or three days the case went on getting worse, ureous vomiting came on, the breath had a distinctly urinous odour, sight was affected; delirium at nights, and a brown-coated tongue indicated the oncome of typhoid symptoms, and the patient's children were summoned. Just then an improvement set in, the urine returned, and the diarrhœa ceased, and the indignant relatives discharged the writer, with scant courtesy, as unworthy of professional confidence. A consciousness of a not having guarded the prognosis sufficiently carefully, would not allow self-pride to assert too confidently that the want of confidence was altogether unjustified. The case proceeded uninterruptedly to a good recovery under the gentleman called in; and the lesson was laid by and pondered over. The next attack occurred some couple of years after, under

another medical man, who held no views as to compensatory actions, and proceeded at once to check the diarrhœa, only too successfully, and this time the depressed balance never swung back, but remained permanently overthrown.

The attacks of apparent enteritis, or colic, ending in copious diarrhœa, the motions soon becoming ammoniacal (W. Roberts), the other serous inflammations, or attacks of bronchitis, carbuncles, or other skin affections, gout, &c., are often recovered from in a surprising manner. But, on the other hand, intercurrent attacks from ordinary outside causes, are more than ordinarily serious, while the effects of variations of temperature, and more especially sudden falls, are very grave. The patient with chronic Bright's disease almost, as it were, lives over a volcano, whose sudden explosions are a source of imminent danger; and these explosions are most commonly induced by sudden falls of temperature, which check the action of the skin, and occasion a sudden accumulation of uneliminated waste in the blood, and from that again some very serious acute affection; the results of which may be unfortunate and destructive to life. Thus a sudden fit of intense cold will throw a number of these cases on the doctor's hands together, and a heavy percentage of deaths among them usually occurs.

Another source of danger is the risk of cerebral hæmorrhage. This is apt to be induced by a paroxysm which will produce arteriole spasm, increased arterial tension, and endanger the elastic tubes which connect the two muscular extremities of the arterial system, already perhaps somewhat rigid and brittle. Thus Kirkes found 14 cases of cerebral apoplexy out of a total of 22, to be accompanied by renal disease, the kidneys, as a rule, being small, hard, and granular, and in 13 of them there was hypertrophy of the left ventricle. And Eulenburg, in 6 cases of apoplexy, found 5 with cirrhosis of the kidneys and heart hypertrophy.

But though hypertrophy of the heart is thus productive of apoplexy occasionally, it is a very important conservative change, endowing the patient with a very fair share of bodily health and activity, as compared to those cases where dilatation exists with very impaired health. For heart failure is

the inevitable fate, whose oncome may be deferred but not averted; and certain and positive increasing degeneration of the circulatory organs must follow this disturbance in the vaso-motor system: an unseen power has laid its arresting hand on the peripheral circulation, whose importance must not be underestimated. There is something weird in the fairy touch on those minute vessels, which is gradually to bring the circulation to a standstill for ever. The condition is not one of precipitation, and much time may elapse, allowing of many an intercurrent accident; ere a point is reached of positive degeneration in the heart itself, which inaugurates a series of backward changes in its turn, implicating the kidneys in common with other organs, and leading inevitably to somatic death. The consideration of the manner in which this heart degeneration is induced, and the consequences which result from it, will engage our attention next.

Part II.

(*From Heart Failure to Secondary Renal Disease.*)

The pathological changes entailed by primary renal disease with accumulation of waste-products in the blood, and their effect on the vaso-motor centre; the consequent spasm of the arterioles, which in its turn led to hypertrophy of the muscular tunics; this obstruction to the blood stream entailing changes in the left ventricle, usually hypertrophy, at first at least; and then the over-distension of the elastic arteries, betwixt the two muscular extremities, have been described above; and now we come to the time when this progress is no longer sustained by compensatory changes.

The distension of the arterial coats, ever existing betwixt the hypertrophied ventricle and arterioles at any time, as testified by the increased bulk of urine, and aggravated at intervals, by the arteriole spasm during a paroxysm, gradually induces structural changes in the arteries. The brittle intima or endarterium with its subserous tissue, becomes the subject of parenchymatous inflammation, and young connective-tissue corpuscles are developed, especially at points subjected to unusual strain. This spreads along from the convexity of

the aorta backwards and forwards. The whole of the larger arteries become affected with this endarteriitis deformans. The growth diminishes, by its own thickness, the calibre of the aorta and its branches: this further taxes the heart-walls.

This low parenchymatous inflammation gradually affects the whole arterial thickness, the tubes lose somewhat of their elasticity and recoil imperfectly after distension; this leads to their being elongated and widened: while their walls become more rigid and less and less elastic. The temporal artery can often be seen in this condition, meandering along the temple, tortuous and serpentine. The arteries are thus not only more liable to rupture, especially during excited action betwixt the struggling ventricle and the spasmodically closed arterioles, but their degeneration affects the heart itself. In the first place the degeneration extends down the coronary vessels, and of course, when so affected, they are not so easily distended by the aortic recoil, their systole; while their elasticity being diminished, the circulation no longer receives the aid it did receive when that elasticity was unimpaired. The aorta itself loses its elasticity also, and its recoil is much more imperfect.

Thus we see that while the heart has to act, or does act, any how, against rigid non-elastic tubes instead of distensible elastic ones, its own nutrition is affected. This no longer enables the ventricle to empty itself perfectly, and imperfect ventricular contraction leads directly to dilatation. The condition is now distinctly worsening, and the blood is still less perfectly depurated, being no longer so freely circulated: this leads to further arteriole spasm, obstruction to the blood stream, and further dilatation. The dilating ventricle no longer possesses the power of the hypertrophied condition, and less blood is thrown at each systole into the aorta, its distension and recoil are less, and the coronary circulation is still further impaired; this involves less perfect nutrition to the muscular structure of the heart, and ultimately structural necrosis, or fatty degeneration results; the worn out material being no longer removed and replaced by new materials from the impairment in the blood supply.

We have now a distinct failure in the heart's power, and distinctly defective circulation. Between the two muscular extremities the heart and arterioles, *i.e.*, the left ventricle and the systemic arterioles, and the right ventricle and the pulmonic arterioles, the equilibrium is deranged, and the defective ventricles no longer counterbalance the peripheral muscular distribution. The impaired aortic recoil alike affects both right and left coronary vessels, and the right ventricle, ever the thinner and more prone to dilatation, yields. This is probably the first step in the congestion of the venous system. The blood, in German phraseology, now lies more on the venous side, and though this soon affects the arterial circulation, the venous congestion remains unrelieved. The venous congestion may obstruct the arterial circulation, and increase the demand on the already over-taxed left ventricle, but, unfortunately, that exercises no curative effect. We have now venous congestion and some new pathological processes entailed therefrom.

In the first place, we get obstruction in the renal vein, and from that albumen and exudation tube-casts: for the venous congestion differs from ligature of the renal veins, or lower vena cava higher up, only in degree. But this is not all, venous congestion affects the nutrient branches of the renal artery, and impedes the flow through them, interfering with the elimination of solids. While these positive results follow from venous congestion, we get, negatively, diminished bulk of urine. This does not result so much from venous congestion, for the narrow continuations of the blood-vessels from the glomeruli prevent its acting very strongly. It is chiefly the result of diminished arterial pressure. Venous congestion means diminished arterial pressure. The blood lying in excess on the venous side, the arteries are not so well filled: this tells on the arterial pressure, which is lowered. We have then diminished bulk of urine.

Lessened arterial tension leads to lowered water elimination from the glomeruli, while venous congestion leads to albumen, fibrin cylinders, or exudation casts, and diminished excretion of urine solids.

A very serious point is reached when the urine becomes

permanently albuminous. The diagnostic import of this sign is significant, and its prognostic import of the gravest character.

This venous congestion leads to still more imperfect depuration, the accumulated waste products cause still further vaso-motor spasm, and thus still further tax the heart, already handicapped most heavily by the obstruction offered by rigid arteries, and the *vis à fronte* of venous congestion; while its structural integrity is impaired. The hypertrophy which once preserved it from dilatation and enabled it to contract perfectly, in spite of the opposition offered, is being cut down irretrievably. The dilatation and structural decay act and react, and ultimately the ventricle halts, the halts become rythmical, then the halts succeed each other; the dilated rotten walls become more distended, and ultimately the heart stops, and all is over.

But this is usually a long and weary process, and the heart struggles away, becoming gradually feebler and fainter. During this time other pathological processes have been inaugurated, and the venous congestion impedes the flow in the portal circulation, being unprovided with and unprotected by valves, and the viscera become subject to growth of interstitial connective tissue, which impairs their functional activity; while the lining of the intestinal canal becomes congested, and often subject to serous flow. The same congestion affects the bronchial veins, and then a serous flow, so-called chronic bronchitis, impedes the patient's breathing and adds to his troubles. The results of venous stagnation have been sketched before, in Chapter IV, and need not now be repeated at length. The brain is now not only fed with impure blood, but it is suffering from the defective arterial flow, while the venous congestion is leading to structural change by connective-tissue growth.

The venous stagnation leads to serous effusions, and these may take place into any of the serous sacs from the ventricles of the brain to the tunica vaginalis, or the fold of Douglass. These add to the circulatory difficulties, and are not unfrequently the immediate cause of death.

Dropsy now shows itself, not purely cardiac, and proceed-

ing from the feet upwards, but more irregularly; indeed, there is a combination of renal and cardiac dropsy. Whether the blood laden with urea does pass more readily into the tissues, than mere ordinary water-laden blood, as is stated by some authorities, or not, is not yet determined. This at least we know, that there is not the same order observed as in uncomplicated cardiac dropsy.

We now get more compound symptoms; dyspepsia from urine salts is complicated with gastric catarrh; attacks of dyspnœa with œdema, chronic bronchial flow, difficult respiration, and emphysema; bilious disturbance comes on; and the sleeplessness of the first stage is complicated by drowsiness and disturbed, unpleasant, and often terrifying dreams. We find the free flow of pale urine of low sp. gr. now no longer. The urinary secretion falls below the normal, and the scanty urine is laden with urine salts. Frerichs on tying the renal vein found the bulk of urine reduced more than the elimination of salts in proportion to each other. But even without experiment this would be manifest; for the blood circulating through the kidneys is overladen (überladen) with histolytic products. Thus the scanty urine is laden with lithates, imperfectly oxidised products of tissue-waste, while it now contains albumen, permanently, and both old granular and new exudation tube-casts. This change in the symptoms derived from the urine cannot, I trust, be a source of difficulty to the reader after the above explanation. The diminished bulk of water from lessened arterial tension, and the albumen and tube casts (new) from venous congestion ought not to create confusion; both depend on failing heart power.

The profuse flow of urine which characterised the first stage of the disease, of the increased tension produced by an hypertrophied left ventricle and hypertrophied arteriole muscular walls, is now modified by heart failure and the lowered arterial tension. The bulk falls gradually as the case goes on, and the urinary flow peculiar to the first stage becomes blended with the changes which follow heart failure, the second stage; until ultimately all trace of the first stage is lost, and the urinary flow assumes the features which charac-

terise heart failure. The urine of the advanced second stage will give no denial to the peculiarities of the first having preceded it, and no diagnosis can be formed from it to reject a previous stage of primary renal disease affecting the heart.

The old granular casts found alongside the new exudation casts will often testify most unmistakably to the fact of a first stage having preceded the condition then present, even if the history be doubtful. The two conditions of first and second stages become blended until the peculiarities of the first are totally lost and merged in those of the second stage. These gradually achieved alterations in the renal secretion, characterising changes in the disease itself, and the grafting of the symptoms of the second stage on those of the first, are well and lucidly described by Sir William Jenner in the Lancet, 1865.

The pathological changes which follow chronic renal disease are well given in these two tables, taken from Rosenstein's "Diseases of the Kidney" (2nd edition, 1870). The one is a large collection made by Frerichs, and the other a smaller collection, from Rosenstein's own experience. The various pathological changes which result from renal disease, uræmic blood, and cardiac failure, are also found to have other affections associated with them; which may possibly enough be mere coincidences, but certainly do create a suspicion of being the result of tissues being fed by a blood not only poisoned with histolytic products, but in many cases deprived of albumen by the constant drain through the kidneys. For many of the cases must have had hearts no longer hypertrophied, but failing; though no cardiac condition is recorded but hypertrophy, simple or complicated, with valvular lesions, the writer's scheme not taking heart failure into consideration.

TABLE of 292 Cases collected by Frerichs.

Kidneys.	Heart.	Lungs.	Pleura.	Pericardium.	Peritoneum.	Liver.	Spleen.	Stomach and Intestinal canal.	Brain.
Stage I. II. III. 20 139 133	99 with hypertrophy. 42 simple. 41 combined with valvular disease.	75 œdema. 4 œdema glottidis. 27 pneumonia. 8 infarctus Laennecii. 3 pulmonary gangrene. 37 tuberculosis. 22 emphysema vesiculare.	35 pleurisy.	13 pericarditis.	33 peritonitis.	26 cirrhosis. 19 fatty degeneration. 1 carcinoma.	26 chronic enlargement. 4 acute enlargement.	24 chronic gastric catarrh. 3 chronic ulcer. 4 carcinoma of the pylorus. 1 typhoid anæmia. 34 hyperæmia and gastric catarrh. 12 tubercular ulceration. 13 follicular ulceration. 2 typhoid ulceration.	11 cerebral hæmorrhage (apoplexia sanguinea). 8 of them with hypertrophy and valvular disease. 2 with atheromatous degeneration. 40 with serous effusion into arachnoid or ventricles. 2 with meningitis. 1 tubercular meningitis. 11 with tumour of the brain.

TABULATED LIST of 114 *Cases collected in the Dantzig Borough Hospital, by Rosenstein.*

Kidneys.	Heart.*	Lungs.	Pleura.	Pericardium.	Peritoneum.	Liver.	Spleen.	Stomach and Intestinal canal.	Brain.
Stage I. II. III. 12 67 35	Hypertrophy in 26. 13 with valvular disease and 13 without. Of these, 9 were in the third, 2 in the second, and 2 in the first stage of kidney disease.	40 with œdema pulmonum. 25 with pneumonia. 3 with abscess in the lungs. 2 with gangrene. 11 emphysema.	22 pleurisy. 25 hydrothorax.	17 pericarditis. 21 hydropericardium.	13 peritonitis.	19 with fatty degeneration. 11 with nutmeg liver. 15 with cirrhosis. 3 amyloid degeneration. 15 simple enlargement (Schwellung).	32 with chronic enlargement. 13 with recent enlargement. 9 cirrhosis. 8 amyloid degeneration.	12 with chronic gastric catarrh. 17 with œdema of the mucus membrane of the stomach and intestines. 16 with follicular catarrh and ulceration of the large intestines. 3 with diphtheria (of the primæ viæ). 13 with catarrh of the small intestines. 9 with follicular ulceration of the small intestines. 1 with tuberculosis of the intestinal canal.	3 with cerebral hæmorrhage. 2 with hypertrophy and 1 with hypertrophy and valvular disease combined. 1 cerebral anæmia. 6 chronic arachnitis. 19 serous effusion into the arachnoid. 14 effusion into the ventricles. 1 purulent meningitis.

* No account of dilatation.

A large proportion of the lesions are almost positively incompatible with the idea of hypertrophy, and must have been results of failing circulation. As regards the combination of heart and kidney disease, these tables prove that out of a total of 406 cases of chronic Bright's disease no less than 125 died during the stage of hypertrophy, a very serious proportion; but there is no record of the heart's condition, where the same causes which produced the hypertrophy must have been in action still in the later stages. It is no more than just to say that of these 406 cases 125 died in the first stage, according to the division adopted here, and that of the 281 cases remaining there is no account as to what was the condition of the heart.

The tables are valuable as indicating the other changes which follow in various organs, some from the attenuated condition of the blood, as hydropericardium and hydrothorax, and others, as pneumonia, pleurisy, pericarditis, &c., from the absolute presence of poisons in the blood; while the remainder, as tuberculosis, follicular ulceration of the bowels, possibly resulted as mere coincidences, or otherwise not admitting of explanation; but probably some of them were the result of the spanæmic blood on which the tissues were fed. The effect of heart failure on each must not be overlooked, especially in the cases of serous effusions, œdema pulmonum, and infarctus. As to the condition of the blood, and its effect in producing or helping to produce the various lesions, there is a very probable connection betwixt the two, and that too of a causational character. In the first place, as to the blood itself, Christison found the normal specific gravity of healthy blood to be 1029 and 1031, but in the subjects of chronic Bright's disease it was as low as 1022. Frerichs has found it as low as 1022 and 1019, and Rosenstein found it as low as 1024. This diminution is attributed to the loss of albumen. Blood so altered and attenuated appears to pass more readily through the walls of the capillaries than healthy blood, even without stagnation in the circulation to aid.

As to the positive contamination of the blood by urinary excreta non-eliminated, Picard found the proportion of urea

in healthy normal blood to be ·016 per cent., but in diffuse nephritis it reached ·070 and ·0846 per cent. In the experiments upon animals of extirpation of the kidneys, ligature of the ureters or blood-vessels, the amount of the various nitrogenised products of histolysis were found to be increased. This accounts for the inflammatory attacks of the serous membranes or lungs found in these tables; while the positive increase in waste-products and the diminution in albumen in the blood account for the other affections from imperfect nutrition. The tables indicate almost to a glance the various intercurrent affections which follow in the wake of chronic kidney (and heart) disease, and which commonly cut off the patient, who is in an already weakened condition. They will also better enable the reader to understand the diagnosis and prognosis of the latter stages of Bright's disease than any amount of other written description possibly could. We have, in short, the objective and subjective symptoms of heart failure engrafted on the symptoms of chronic renal disease described in the first part of this chapter. The changes in symptomatology entailed by this have also been alluded to in this section, and each individual case must explain itself and its symptoms, for to go over all again now would be a needless repetition or recapitulation.

The progress and termination of these cases is as unpleasant a subject for contemplation as medicine furnishes, for sudden death alone is to be hoped for. Without it, the progress is steadily downward with the vicious circle ever widening, and new miseries added to the load, which the patient has already found to be wearily intolerable. Difficult respiration, utter exhaustion, with sleep even more horrible than waking, with terrifying dreams taking the place of waking horrors; the prospect is utterly unrelieved by a single ray of hope other than speedy dissolution, by sudden death or intercurrent inflammation. While the future contains no hope, retrospection is embittered by the long list of troubles endured, of miseries survived. The long vista, which the patient's memory furnishes to him of what he has existed through and suffered, since first disease in a depurating organ led to chemical changes, entailing mechanical results

in their turn, is merely an avenue of tortures, steadily increasing as time rolls; the first ones nearly imperceptible in the distance, the immediate ones looming large and near. The gradual oncome of new additions is often extended over a period of many years, each new addition marking an epoch, on entering into another stage worse than the one preceding it, until the past condition, deemed bad enough at the time, is looked back to almost with a feeling of envy; and its troubles would be thankfully accepted in lieu of the present ones, were such retrograde movement practicable. The condition differs but in degree from Dante's Hell, or the sketches of future punishment in monkish writings, where as soon as habitude has rendered one condition of misery somewhat less intolerable to the damned, another is supposited more fearful still, with the additional scourge of novelty. The patients themselves give utterance to this, often saying, "this new ailment frightens me so; for I had got accustomed to the old one." The brain, fed with blood robbed of its albumen and poisoned with urine salts, and even that but sparingly supplied to it by a failing heart and rigid arteries, and oppressed by venous congestion, falters, and its manifestations become imperfect; the mind no longer preserves its accustomed tone; the will is sapped, and the intellect is enfeebled and becomes childish; no longer capable of engaging itself on other matters, the attention becomes centred on the physical condition alone; the world shrinks to the dimensions of the sick room, and the patient gradually passes away from the world and its interests; and when, at last, all is over, the sensation is rather of trouble removed, of relief experienced, than of loss sustained.

Treatment.—The foregoing description of the pathological changes and their symptoms will have prepared the reader for the announcement that the treatment of this condition is far from a simple matter. At first it is not so difficult, but, as the case progresses, the gradual involving of some previously unaffected organ and the addition of a new evil, the gradual worsening generally; with the various compensatory actions, each a subject for congratulation, if not excessive, but easily becoming a source of great danger, present a

problem not to be solved lightly, and often taxing to the utmost the physician's skill and resources. For the treatment becomes in time nearly as complex as the disease, and the remote consequences of our measures must be taken into consideration, and possible contingencies set against immediate relief to be obtained On the first recognition of the disease, there are two great considerations and lines of treatment to be adopted. 1. To preserve the purity of the blood itself; and 2, to guide the various morbid steps so as to secure a maximum of compensation, and a minimum of danger resulting therefrom.

To preserve the Purity of the Blood.—This is a matter of great moment, and there are two opposite conditions to be fulfilled, viz., to prevent accumulation of waste on one hand, and to avert anæmia on the other. We will consider the prevention of waste first. We know that an exclusively nitrogenised diet increases the amount of urea and uric acid, and, of course, these passed through the earlier stages of creatine, creatinine, &c. This accumulation of waste must be obviated by exact diet, and the amount as well as quantity must be laid down. Any excess of nitrogenised food would add to that condition called "uræmic." In speaking of this condition, Dr. Bristowe says, in his Croonian Lectures on Disease and its Medical Treatment, 1872, "I may add that it would seem theoretically, and I think that it is, of the utmost importance, in treating uræmic cases, to take measures to check the formation of urea in the system; and that to this end the diet should be carefully regulated, and not be allowed in quantity to exceed the actual requirements of the system." This is the first point to be attended to, namely, to secure, as far as possible, a nice adjustment betwixt the needs of the system, its powers of elimination, and the amount and quantity of food consumed. Some systems assimilate a much greater proportion of the food taken than others do; this must be allowed for, and an analysis of the urine and a pair of scales for the food will scarcely cover the necessities of the case. Even the effect of a large consumption of hydrocarbons in arresting the oxidation of azotised matters must not be lost sight of here.

Then there is the second half of this matter, the securing as complete depuration as is possible. Potash, lithia, and the waters of Carlsbad or Vichy, and biphosphate of soda according to Ritter von Schroff, the Professor of Materia Medica at Vienna, will affect the accumulation of uric acid, and Dr. Roberts of Manchester, speaks in favour of citrate of potash, and with every reason, in my experience. The effect of sub-acid wines in checking the elimination of uric acid is undeniable; the advertisements of wine-merchants to the contrary notwithstanding.

Iodide of potassium is a good adjunct often, and colchicum, buchu, juniper, apparently act by increasing the flow through the nutrient vessels, and the elimination of urine solids. Arsenic, as Fowler's solution, may often be added to potash advantageously, especially where the skin is inactive. Mercury should be avoided from the known intolerance of it in chronic renal disease. Laxatives, and general attention to the bowels are important; and baths and a good action on the skin are clearly indicated. Fresh air and exercise are good, and aid in oxidising the waste products lingering in their decomposition, and reduce the necessity for rendering uric acid, or, as it must exist in the alkaline blood, in union with some base, its insoluble compounds, soluble by potash or lithia. In administering alkalies, it must be borne in mind to give them when the stomach is alkaline and empty, and not with food, or after it, when the stomach is acid; except in those cases of acid gouty dyspepsia, where the excess of acid may be advantageously neutralised. The use of the alkaline solvents of uric acid must be continuous in small doses to meet the continuous formation of the acid itself; and not in intermittent large doses, unless in paroxysms or exacerbations, when alkaline laxatives are indicated. The bottle of granular citrate of potash on the dressing table, always at hand, for a tumblerful of water and a teaspoonful of the salt, on first getting out of bed, or waking in the morning, and again when commencing to dress for dinner, is a very good and palatable means of checking the accumulation of uric acid, and, of course, its consequential results. This is even better than potash-water

with or without gin, though that is a therapeutic measure not to be sneered at. Potash has a decided tendency to act on the skin, much more indeed than commonly thought, and is thus doubly useful.

In other cases there is a decided tendency towards anæmia. This effect is probably due to the presence of the excreta in excess, amounting to a blood poison, an action which it shares with malarial poison, syphilis, lead, &c. There is a decided tendency in these blood poisons to anæmia and consequent neuralgiæ, and the treatment must not only contain remedies more or less specific in each case, but also the administration of iron and other hæmatics. Iron and quinine in combination with strychnine are often of great service. What effect they exercise over these arterioles, here hypertrophied, is not yet known, but they seem to dilate the unchanged minute vessels of the cerebro-spinal system at least. Iron is especially desirable as aiding in the formation of red corpuscles, which are diminished here (Rosenstein), in company with other conditions of blood-poisoning. It is often best given after food, and is then digested with the food, in the shape of drops, pill, or powder. This effect is much aided by some easily assimilable fat, as cod-liver oil, and this combination is very useful in many cases where the tendency is to anæmia. Why, it is difficult to say; but chronic kidney disease in Germany has always conveyed to the writer the very strong impression of assuming an anæmic type rather than a gouty one, the reverse being, rather, true in England, even with the patients of a public Institution. Women are unquestionably more liable to put on the anæmic than the gouty type, and the same may be said of the younger subjects of this chronic affection. Whether it has anything to do with the morbid process and its progress in the kidney, or not, cannot be settled dogmatically either way. The small granular kidney is apparently less associated with loss of albumen by the urine than other forms of disease. All idea of affecting the progress of the disease in the kidneys by anti-inflammatory agents cannot be seriously entertained in the present state of our knowledge, and parenchymatous inflammations are all very heedless of antiphlogistics. The

prevention of further disease by the avoidance of accumulations of histolytic products, and especially that form in which the imperfect oxidation is most liable to be persistent, namely uric acid, must be avoided; as there does exist some ground for the suspecting of uric acid of acting as a tissue-irritant, much like alcohol. Debauches are obviously to be avoided, and those results of internal congestion of the viscera from general contraction of the dermal capillaries induced by exposure to cold or draughts, must also be studiously avoided; for the subjects of chronic Bright's disease are as susceptible to atmospheric changes as a thermometer or barometer. This extreme susceptibility is insisted on by all writers on renal disease, and the patient must be clothed with flannel or silk next the skin, or submit to the awkward consequences of neglect. They should ever have a top-coat when stepping out of doors, and imitate the Austrians, who are very particular about that point, if not remarkable for attention to other matters of hygiene.

We will now consider more particularly the effects on the circulation, and the means by which this effect may be minimised. The first step was vaso-motor spasm, by the effect on the vaso-motor centre according to Ludwig and Traube, and the production of hypertrophy of the muscular tunics of the arterioles, as shown by Johnson and others.

Now it is very obvious that this effect produced by retained tissue-waste circulating in excess must be almost constant to a greater or less extent; and that there will be times of less impurity than others.

The effect of this arteriole spasm is to check waste, it is quite true, and thus prevent the blood from excessive poisoning by reducing the waste to the power of the kidneys. This conservative action, however, is followed by results on the left ventricle, and, according to Traube, before any very long time elapses. It is obvious that the first step in treatment consists of reducing the impurity to a normal minimum, as far as possible, by the means mentioned above. A paroxysm of disturbed renal innervation leads to excess of water and diminution of salts in the urine.* There is then an exacer-

* This view of the antagonism betwixt renal flow and amount of solids

bation of symptoms, as may readily be conceived; we have now a precisely similar train of events from the cause being present in excess. Catharsis, or profuse diaphoresis by hot-air bath, or otherwise, are indicated. During this vaso-motor spasm we find very often palpitation, as an evidence of heart taxation. The obstruction to the flow of the blood in the arterial system tends to prevent the complete contraction of the left ventricle, and this imperfect emptying leads to dilatation. But very commonly, according to even the imperfect tables (imperfect in this question of effect upon the heart at least, for probably in the other cases the hypertrophy had yielded to dilatation, or dilatation had always existed), the impression of distension excites, though the vaso-inhibitory nerves, a freer circulation through the coronary vessels, and this leads to hypertrophy, which resists the distension, and also enables the heart to perfectly contract.

Dilatation in the heart is a very serious matter here, as any imperfection in the heart's power would lead to venous congestion, and then we would have an effect upon the kidneys produced therefrom, and still further imperfect depuration. It is obvious, then, that in the treatment of this condition we must rely strongly on agents which increase ventricular contraction, of which first stands digitalis, and after it belladonna, caffeine, squill, &c. Especially in the paroxysm must digitalis be used when an additional obstruction (spasm) is added to the permanent condition (hypertrophied arterioles). It is true that digitalis does affect the arterioles, or capillaries, for Hughes Bennett, Stricker, and others, think the capillaries contractile, probably as part of the vaso-motor system, which is affected by it in increasing the contractions of the heart itself; but still its effect upon the heart is most important, and the arteriole effect must be met by depurating the blood, as thoroughly as is possible. No fear of the effects upon the arterioles must deter us from

receives a singular corroboration from Traube, Niemeyer, and others. They point out the small bulk of urine and high specific gravity in amyloid kidney as a diagnostic sign, distinguishing it from chronic Bright's disease. In amyloid disease the glomeruli are most affected, and the tubes comparatively normal, and the diseased glomeruli obstruct the flow in the convolute vessels, and direct the blood rather to the nutrient branches.

seeing the dire results which will ensue from dilatation of the ventricular chamber, with all the train of evils which result therefrom. The combination of digitalis here with potash, colchicum, and buchu, is of great service; and even the combination without digitalis will often affect palpitation, thus occurring, better than will digitalis, without the other agents, which assist so markedly in producing an increase of solids in the urine. There seems some reason for supposing that agents which produce increased elimination of urine solids, do so by virtue of some effect over the innervation of the kidney, *i.e.*, they probably produce dilatation of the nutrient branches of the renal artery. Some experiments are much needed here to show the antagonism betwixt water elimination and urine solids: it is obvious enough that if such an effect is produced on the nutrient vessels by attracting a larger portion of blood from the renal artery, the pressure in the convolute branches must be lowered. At least we already know enough to be conscious of the relief which follows their administration in this condition. One great difficulty to be surmounted in the treatment of chronic renal disease is the constant tendency for the urine excreta to assume the form of uric acid, which is very persistent. This arises probably from the impaired action of the kidneys, which no longer convert the earlier products of retrograde tissue metamorphosis into urea, a readily soluble salt passing readily out by the kidneys. Injections of urea into the blood have not produced uræmic head symptoms, and the chemical results of an exacerbation are, in all probability, imperfect conversion of the earlier products into urea or even uric acid. For these earlier products are by far the most fatal to life, and most quickly fatal if we compare the results of ligature of the ureters with extirpation of the kidneys; and their conversion into uric acid even is a gain, for it lessens the immediate danger, if it does ensure structural changes in the future. In the treatment of uræmia acute catharsis is our chief hope, and diaphoresis is a great adjunct; the Germans approve of cold to the head, but on what hypothesis I have been unable to ascertain. B. W. Richardson in his essay on uræmic coma (Asclepiad) gives a

case of uræmic coma simulating apoplexy which must not be omitted; for it is most instructive as well as well told.

General venesection is positively of service, in relieving the convulsions at least, and reducing the symptoms. How it is achieved is not yet to be explained. The uræmic convulsion and coma may to some extent be regarded as a peroxidation, as a useful action, for Rosenstein states, "The temperature is, so I have found, distinctly raised during the uræmic attack, more during the convulsions, but also during the coma," p. 152. That it is a form of peroxidation of a very alarming and too frequently fatal character, is only too well established.

Compensatory Actions.—This now brings us to that division of treatment which more especially belongs to the plan of treatment of the acute attacks in other organs, which so frequently occur during the course of chronic kidney disease. These are frequently compensatory actions, as diarrhœa, vomiting, acute inflammatory attacks, &c. They are often preceded by certain peculiar symptons supposed to indicate acute congestion of the kidney. These consist of lumbar pain, weight over the loins, malaise, scanty urine, often approaching almost complete suppression, and albumen or blood itself in the urine. Then comes on a pretty smart action in some other part, very often acute diarrhœa. Now it is very obvious that here we have a compensatory overaction, and not a morbid process to deal with; though it is not to be questioned that the action may become a source of danger to life, indeed these patients are not uncommonly cut off by these intercurrent attacks.

But it is very certain that a restoration of the action of the kidney is the most important point in the treatment.

Thus cupping, wet or dry, over the loins, or hot poultices kept on continuously, and night and morning faced with mustard, hot-air baths, where practicable, and some non-irritating diuretic as potus imperialis, or potash and buchu may be given. On the restoration of the renal flow, the diarrhœa will usually subside spontaneously.

All opiate remedies are unsuited to this condition, and the combination of opium with vegetable astringents is calcu-

lated to arrest that recovery within the kidney, and restoration of its secretion, which we are so anxious to secure. When the diarrhœa is becoming itself serious, and assuming an attitude which makes it a source of danger to life, and some treatment of it is absolutely called for, a combination of potash nitrate, pernitrate of iron, and calumba has appeared to me, in practice, to be a good remedial agent against the diarrhœa, and with a minimum of action on the kidney. When the renal flow is established, and the diarrhœa persists, more powerful astringents may be indicated; but it must not be forgotten that opium, like mercury, is unsuited to chronic renal disease, which seems to endow the patient with great intolerance of these famous members of the pharmacopœia.

Vomiting here is somewhat difficult to treat, in consequence of the remedies being rejected by it. It is regarded as a serious indication, usually only occurring in grave conditions. It may be met by cathartic injections per rectum, and the hot air bath and local applications to the loins. Dr. Charlton, of Newcastle, says that in uræmic vomiting nothing is so effective in checking it as carbolic acid in small doses largely diluted. This may sometimes be indicated from the severity of the vomiting, but the restoration of the natural secretion is certainly the first indication.

Intercurrent Inflammatory Attacks.—When these occur in an organ scarcely to be regarded as an eliminating organ, as in the serous membranes, even then due regard to the cause will be of great service, and cathartics, local applications to the loins, and mild diuretics will be useful. How the lungs stand, and how far pneumonia can be regarded as an eliminant action of the lung tissue, may not be readily settled. Still, when pneumonia is dependent on a poisoned condition of the blood, it is better to treat the blood than the pneumonia, and one case, made a very vivid impression on me, where pneumonia (right side) came on in an old friend, who had had more or less constant hæmaturia for thirty years, and whose urine at the time looked very like blood; but he asserted it was better than usual, and so confirmed my impression of the attack being uræmic. Potash,

juniper, and buchu constituted a very rapid and satisfactory curative treatment, and the patient, who had been troubled by a teasing cough, as a symptom of the lung affection, gave his opinion very confidently to the effect that it was "the best cough mixture he had ever taken." These inflammatory affections are better left alone than rashly treated, and there is little room for doubt that such cases have often been appealed to as brilliant successes by the advocates of an expectant treatment, the believers in an alkaline treatment of inflammations, and other practitioners, denominated irregular. The writer cannot shake off the impression that the foundations of the success of the alkaline treatment of acute inflammations are planted on these uræmic acute affections. Each affection occurring as an acute intercurrent ailment in the subject of chronic Bright's disease, must be specially investigated as to how far it resembles similar affections in healthy persons, and to be treated accordingly; or to be due to the condition of the blood. If the affection is recurrent, or even if the patient be subject to other affections, and have repeated attacks of illness, it creates a suspicion of the nature of the malady and of its connection with chronic renal disease, even if such disease has not hitherto been suspected.

Albuminuria.—In many cases of chronic renal disease the patient is subject to recurrent losses of albumen, on a very large scale, and sufficient to impoverish the blood very markedly. Here the administration of tannin in large doses, or gallic acid, has been advocated, and the loss met by eggs and other albuminous articles of diet. This loss of albumen is more apt to occur in the anæmic forms than in the gouty type of renal disease. This anæmic type, as said before, is very common in Germany. Still Niemeyer is not in favour of large doses of tannin. Frerichs advocates its use, however, and has a favourite form in pill with the watery extract of aloes. A. Hill Hassell and others in England advocate tannin and gallic acid, while Beale and others advocate preparations of iron, and especially the perchloride. The latter is perhaps, on the whole, the better plan of the two, as the iron is a more decided hæmatic than tannin. The effect of

anæmia in weakening the heart must not be overlooked in its treatment. Before leaving the treatment of the first stage, it may be desirable to mention that the attacks of apoplexy occurring in this stage do not often admit of relief, and are very commonly fatal. Free venesection and catharsis are here indicated.

Sleeplessness.—Sometimes the patient will seek medical aid for the sleeplessness of this condition. Opium in all forms is most unsuitable, even in the form of the popular subcutaneous injection. It is not well borne in these chronic renal conditions, often not only failing to produce sleep, but even occasioning greater wakefulness, with great mental disturbance. It certainly affects the kidneys, whether by arresting the action in the epithelium cells of the uriniferous tubules, or how, is not yet ascertained. Opium and morphia are often resorted to for this very action, of arresting the elimination of urine solids, in the condition of azoturia, or excessive excretion of urea. Some of the other "seven sisters of sleep," as hyoscyamus, belladonna, tincture of hop, lactucine, &c., may be used to combat this sleeplessness, or the now popular remedy, hydrate of chloral.

Treatment of the 2nd Stage.—This is eminently unsatisfactory and is ever palliative rather than curative. It is the treatment of failing heart modified by renal complication; that is, it is a form of heart failure which goes very readily downwards in spite of all treatment, when once the heart failure is marked. When the hypertrophy is being cut down by fatty degeneration, by molecular necrosis within the muscular primitive bundles, all treatment of the heart by digitalis, iron, &c., is devoid of any lasting value. There may be a fair amount of muscular structure remaining sound, and thus giving out good first sound, and palpitating vigorously at times, especially on exertion, but the condition is not a remediable one. It is not mere failure of the heart, it is failure before a blood current, obstructed by hypertrophied arterioles; and spasm induced by blood still less depurated from the effect of venous congestion of the kidneys, and accumulation of waste products in the blood, even more than before; of failure combined with a rigid non-elastic aorta,

with its feeble recoil met by spasm in the arterioles of the coronary circulation; is it any matter for surprise, then, that drugs are ineffective here? It would be matter for much greater surprise if they were effective under such circumstances: and those who find digitalis in some cases apparently inert, are seriously recommended to go over the case again more thoroughly, to ascertain, beyond doubt, how far the failure lies in a diagnosis itself imperfect, and not taking in all the factors, and not in any inertness in the agent.

These cases go rapidly downwards, and are little, if at all, amenable to treatment, other than action on the various depurating organs; relief of attacks of dyspnœa by hot applications to dilate the minute vessels of the skin, and even, at times, by venesection when the danger is imminent. Free purgation, by relieving the congestion in the valveless portal circulation, and this affects the renal veins, will not often only give immediate relief, but even does good for some little time after, by the improvement in the circulation within the kidney, thus admitting of the blood being brought more freely in contact with the depurating organs, as well as increased elimination of water.

In one point, however, the dropsy of this combined condition differs from that of pure heart disease, and that is its being better relieved by incisions or punctures. There are some points of difference in its production, and the thinning of the blood modifies the stagnation, and dropsy will occur in a less feeble condition of the heart than when renal disease is wanting. The loss of albumen and positive presence of salts, and tissue waste in excess, appear to cause it to pass more readily into the tissues It gives relief to the circulation often, and patients recover and go out again to resume their occupations in a manner different to pure cardiac dropsy. In fact the earlier attacks may fairly be considered renal dropsy, little, if at all, modified by the heart affection.

But when the heart failure becomes pronounced, this renal character becomes merged into the dropsy more especially cardiac, and though it never is so strictly an advance from the most dependant parts upwards, and is found in the upper

extremities and face, more than in true cardiac dropsy, the condition becomes practically cardiac.

The treatment depends on the condition: if the heart's action be good, hot-air baths, diaphoretics, and cathartics, especially such as pulv. scam. co., gamboge with bitartrate of potash, elaterium, &c., are indicated; and incisions may be useful. Digitalis, squill, &c., so useful as diuretics in heart failure, are not indicated here, though very serviceable after the heart's action, tells us it is failing in power, and unequal to maintaining fair arterial tension.

In all complications the presence of renal products, and more especially the most permanent form of uric acid or urates, must not be overlooked in the treatment, and potash assumes a much more important position in our armentaria than it does in simple heart failure. Thus the dyspepsia is gouty as well as catarrhal; so also the bronchitis; the skin is much more liable to affections, and especially gouty eczema. Alkaline lotions and the internal administration of alkalies is indicated. The waters of Carlsbad, Vichy, Vals, Seltzer, Kissengen, and other alkaline-salt waters are here indicated, and are easily procured now in bottle; or potash water, and potus imperialis.

The other alkaline chalybeate waters, as Pullna, Pyrmont, Schwallbach, &c., and the bitter waters of Fredericshall, rather belong to the first stage, but are often in place even later on. The free dilution of the alkalies administered in combination with other agents, from the medicine bottle, is advisable, and then they resemble in effectiveness more the natural waters.

The general treatment of this stage must be conducted according to the rules laid down in the Chapter on the Treatment of Diseases of the Heart, with the modifications indicated above, and such others as the necessities of each individual case seem to indicate.

Kidney Disease the Result, and not the Cause, of Heart Disease.—Early in the observations made as to the connection of heart and kidney disease, one set of authorities maintained that the kidney disease stood to the heart disease as a consequence, and not a cause. But in time this view

got, to a great extent, lost sight of, and for some time past kidney disease has rather been regarded as the cause of heart disease, than as the result of it. Having considered kidney disease in relation to heart disease as a cause, and also the effect of heart failure back again upon the kidneys, the writer now proposes to consider kidney disease solely as a result of heart disease. When from any cause, but usually acute injury to a valve, as from rheumatic fever, &c., the heart is injured, so that its function is imperfectly carried out, there results a certain amount of venous congestion, from the blood lying more on the venous than the arterial side. There follows from this plethora abdominalis, or venous fulness of the abdomen; this venous stagnation being most felt in the valveless portal circulation.

The venous fulness is most apparent in the divisions of the lower vena cava in heart failure, and the veins of the lower extremities being protected by valves, this venous stagnation affects most the portal veins, not being so protected. Thus we find that the various viscera, as liver, spleen, intestinal canal, kidneys, and generative organs, are subjected to an impeded circulation from this venous congestion. The same impeded circulation also affects the lungs and cerebro-spinal system, as we saw in Chapter IV. This impeded circulation leads to a peculiar form of structural change in the viscera. The higher tissues, or higher histological forms, are not affected, but the connective-tissue, or basement-membrane, that is the lowest histological form out of which the higher textures are developed, becomes affected by this vascular repletion. This connective-tissue is found, more or less sparingly, in every viscus, and lies betwixt the higher tissues, occupying the interspaces and forming a sort of packing. When there is venous congestion, or venous hyperæmia, there is a proliferation of cells in this low histological form. The organs become somewhat larger, denser, and on section a glutinous bloody fluid exudes; in time a distinct cirrhosis results, and we get cirrhosed liver (nutmeg-liver) from the shrinking of the new connective-tissue growth, and a similar condition of spleen, the organs being hard, firm, and contracted. There is in organs permitting of it, an

accumulation of albuminous fluid, as ascites, hydrocele, &c., and from the mucous surface a flow of albuminous fluid. The kidneys are also involved, and their scanty connective-tissue becomes the subject of hyperplasia from venous hyperæmia. The kidneys, after a valvular affection of the heart, proving quickly fatal, are somewhat enlarged, vascular, deep-coloured, and injected; a bloody fluid exudes on section, and to the touch they feel finer and denser than the norm: in fact, instead of a mass of vessels and tubules with very scanty connective-tissue, there is now found a distinct growth of new connective-tissue. The capsule is still smooth, but either generally deep reddish, or the surface is chequered with stellate injection of the venules (stellulæ Verheynii), the cortical substance is thickened, injected in streaks, or of a greyish red colour; the cones are hyperæmic and darkened, while the papillæ are pale in comparison.

The epithelium cells are coiled up (gewundenen), and the tubules filled with blood-tube casts, or stained albuminous masses, occasionally undergoing fatty degeneration. This account of von Dusch is perfectly corroborated by observations made by the writer in the Pathological Institute of Vienna during the past winter; when the kidneys, along with the liver and spleen, were found altered by heart disease, with the certainty of a physical law. When the kidneys were long affected by old-standing heart disease, they were small, hard, the cortical substance small and atrophied, with the surface granular, and the capsule adherent; in fact, they were cirrhosed kidneys.

This interstitial nephritis, for it is a parenchymatous inflammation according to Virchow, may also be accompanied by metastatic nephritis from embolism, but usually is simple and uncomplicated. It arises from venous hyperæmia exactly as does the same interstitial disease of the liver, spleen, &c.* This affection of the kidneys from heart-disease,

* From the accumulations in serous cavities, from venous hyperæmia, the albuminous urine, and albuminous discharge from the bowels, it would seem that in venous hyperæmia there is exudation of liquor sanguinis, either from surfaces or into tissues; and that in the tissues the excessive supply of albumen leads to cell-proliferation in the connective tissue, and this leads ultimately to compres-

especially valvular failure, but equally from any other cause leading to venous congestion, has been more recently investigated by Förster, Oppolzer, Traube, Rosenstein, and von Dusch.

In 67 cases of granular-atrophy of the kidneys, Förster found no less than 26 with valvular disease of the heart; and came to the conclusion that valve-failure was a cause of chronic Bright's disease. Oppolzer regarded chronic renal disease as a consequence of valvular disease. Traube has held this view strongly, and found the symptoms of this renal disease, as given from the renal secretion, to be diminution of bulk (from loss of arterial pressure), and a consequent concentration of the urine, with deposit of urates on cooling, the urine more or less albuminous, from "transuded blood-serum" (transudirt blutserum), and with long, hyaline, exudation-casts (schlauchförmige Cylinder), in fact just what Frerichs got from ligature of the renal veins. Rosenstein has taken up a modified position, and regards chronic kidney disease as sometimes the consequence, and at other times the cause, of endocardial disease. These different observers all agree in the view of heart failure, valvular or other, being a cause of interstitial nephritis; and this view was taught in Rokitansky's school. The recognition of the fact that structural changes in the kidney follow heart failure is an important matter, not only from a prognostic, but from a therapeutic point of view.

When the course of the heart failure has been short, the changes in the kidney are evidently of recent date, but where a heart lesion has existed some considerable time, the kidney disease is of longer standing, and more gradual. An acute failure, sufficiently marked to cause death in a few months, causes a general interstitial cell-proliferation in the kidneys; but none the less does the process go on in the more chronic cases of slight valvular affection, not enough to kill, but only to cripple. Here there is a venous congestion less marked but enduring, the same process only extending over a longer period of time. Thus in these cases the original valve lesion is met by compensatory muscular growth, and the compen-

sion of the other structures, first from simple pressure, and later on by compression from the contracting or shrinking of the new tissue.

sation is long maintained; but still no compensation is entirely perfect, and so the impediment in the circulation slowly and insidiously lays the foundation of a new obstructing process by the installation of chronic renal disease. Then comes the circle of mischief detailed in the first part of the chapter, the vaso-motor spasm, with hypertrophy of the arterioles, and obstruction to the blood stream; this, in addition to the old valve lesion, brings about dilatation and heart failure; and the valvular lesion, years after its inauguration and apparent compensation, leads ultimately to heart failure round by the kidneys. This effect of venous stagnation on the kidneys, of structural changes induced by venous hyperæmia, points out how very important it is to secure good acting power in the heart, not only for its own maintenance, but also to avoid, and delay as long as possible, the venous congestion which inaugurates the renal changes. The importance of securing good ventricular contraction, of good circulation, is demonstrated unanswerably, and the therapeutic importance of that list of agents which induce increased ventricular contraction is enhanced. We have seen that heart failure is the turning point in the progress and prognosis of chronic renal disease, when a cause of heart disease; and now we see what a disaster impeded circulation from heart failure is in ultimately overtaxing the organ already enfeebled. The clinical importance of avoiding venous congestion from heart failure, of improving the action of the heart by aiding in securing compensatory hypertrophy, of delaying, as long as may be, the venous congestion, no longer to be averted, is demonstrated only too clearly. In heart disease the appearance of albumen in the urine, with exudation-casts, marks the inauguration of a secondary process which will work evil, and that, too, in no long time. In a little while a chronic Bright's disease is firmly established with all its consequences, and it may not be possible always to determine which lesion came first in the order of time; they are there together, and the combined condition of heart and kidney disease is complicated by a lesion in the heart itself, whose effect upon the case and its prognosis is the reverse of desirable.

The valve lesion may have inaugurated the renal disease,

or the renal disease may have led to the valve lesion, for endocarditis is a common consequential complication of chronic Bright's disease; at any rate they exist together, and the valve lesion is accompanied by granular tube-casts, &c., and the symptoms of chronic renal disease. In the same way kidneys are found, which, while presenting the evidences of general recent interstitial inflammation, carry on their surface pits, or scars, old depressions indicating a primary renal disease, which has led to changes ultimately entailing heart failure, and the implantation of recent disease on the old primary affection. The mutual effect of heart disease on kidney disease, and the causational relationship of chronic Bright's disease to heart failure, are now sufficiently thoroughly established, and admit no longer of doubt as to their pathological connection; and the sooner an equally complete recognition of this connection is entered in our therapeutical indications the better.

If we do possess agents which increase ventricular contraction, and thus maintain a more even balance betwixt the amount of blood in the arterial and venous system, without allowing that balance to found by capillary congestion (venous hyperæmia), it is time that the importance of that action be fully recognized. And whatever vestiges of old theories of accumulative actions, and of dangers arising from an administration of the drug in the wrong condition, may linger; it is quite time that these declining ideas be either laid quietly in the tomb or their absolute accuracy be demonstrated, and rules founded thereon and framed thereby, be laid down as to the action of digitalis and other agents increasing ventricular contraction. For as to the benefits to accrue from their use, and the dangers to be positively averted or delayed by their administration, there is no question. Let, then, the shadowy dangers, said to arise during the use of digitalis, be fairly set against these benefits and a balance struck. That the morbid downward process can only be delayed, in the great majority of cases, by treatment it is true; and that increased ventricular action will ensure arterial distension and atheroma is equally certain; but all treatment is the substituting of a lesser for a greater evil; and with the positive danger of venous congestion and

consequences looming large and distinct, we cannot wait in our action or delay our treatment, except for very valid reasons shown why it should not be proceeded with.

The falling off in the bulk of urine is ever an indication of an unpleasant prognostic character, as indicating decrease in arterial tension; while the positive presence of albumen and exudation-casts in the urine are signs, whose import is only too unmistakable, in the course of a heart lesion. The heart, already somewhat incompetent, will soon have another very serious addition to its troubles in the shape of arteriole obstruction; and if any treatment can restore, however partially, for a time the balance betwixt the amount of blood in the arterial and venous system, there is no time to be lost in applying it.

This chapter has grown somewhat lengthy, but the importance of the subject must excuse that. For, in the present age, when heart disease is increasing, induced by the rate at which we live; when the great frequency of chronic renal disease is being understood; and finally, when pathologists are demonstrating how these diseases act and react; the practical physician cannot overlook the importance of the connection nor its therapeutical indications. We have seen how a slight affection of the kidney will disturb the balance betwixt the material to be excreted and the power to so excrete it; how this will lead to ultimate degeneration in the circulation, and secondary renal disease therefrom, ere it is ultimately fatal. We have also seen how a valvular lesion of the heart, by disturbing the balance of the circulation, may lead to renal implication, and from that produce disease which admits no longer of compensation; how the first compensation by muscular growth is in time destroyed by another call upon it. We have seen enough of the mutual relationship of heart and kidney disease to see that there can be no complete comprehension of the one, possible even, without a fair understanding of the other, and any defects in this chapter must have allowances made for them; while the writer trusts that this attempt to sytematically arrange this connection, with the results arising therefrom, will lead to more perfect and elaborate working out of the subject by other writers.

CHAPTER XIV.

Diseases of the Great Vessels near the heart.

(This chapter is absolutely necessary, as many affections of the vessels in its neighbourhood are very similar to disease of the heart itself in symptoms, signs, and course. It is only in so far that these affections are considered here; and aneurisms of the arch and descending theoractic aorta, together with abdominal aneurisms, are not part of the writer's plan. Aneurisms of the ascending aorta, or of such portion only as is covered by pericardium, simulate heart-disease itself closely, while, when in other parts, they have other signs or symptoms which point to their aneurismal character).

The Atheromatous Process—Aortic Dilatation—Aneurism—Symptoms—Signs—Prognosis—Treatment.

Atheroma is the name in common use in England to signify disease and degeneration of the arteries, but its synonyms endarteriitis deformans, seu nodosa, or arterio-sclerosis are also in common use elsewhere. The disease is not due to any acute inflammatory action in the arterial coats, a disease whose existence is disputed, but is one of the interstitial parenchymatous inflammations of Virchow, and consists of a proliferation of connective-tissue elements under the tunica intima. The first appearance of atheroma consists in localised patches of these elements forming a papule under the intima and often placed at points subjected to great tension; indeed that is its favourite seat when localized. At other times this proliferation goes on in the sub-endarterial tissue in a more general manner, and gives a roughness and variegation to the artery when seen opened, the arteries appearing as if small rice-like bodies were inserted beneath the tunica intima.

The new growth itself consists of masses of young connective-tissue, with small, round, and spindle-shaped cells, and when in localized masses mucin is also found, giving the mass the appearance of slime. When the affection is more diffused the small masses are firm, solid, and resistant to pressure.

This chronic inflammatory process becomes modified in two directions during its progress, each a degeneration; these two are calcification and fatty degeneration.

Calcification, or petrifaction, the latter often the more applicable term, consists of the deposit of earthy salts in these masses of connective-tissue. Sometimes the mass is somewhat crumbling and mortar-like, but at other times, and especially near the aortic valves, the process is rather to be denominated petrifaction, a stony feel being given to the touch. This process depends on the deposit, or infiltration, of lime salts into the connective-tissue corpuscles, and is a rude attempt at ossification, the term used by older writers. This will proceed, when in patches, until a stony plate is formed, which may ultimately become loose, and wash off into the circulation. When the affection is more general, the arteries become converted into rigid brittle tubes, often so impairing the circulation through them as to lead to gangrene of the extremities, especially the lower.

Fatty Degeneration.—At other times these collections of young tissue undergo fatty degeneration, become softened and yellow, consisting of fatty granulations, or oil-drops even, with cholesterine scales, and morsels of connective-tissue, forming what is called a purèe of pease. This mass may become washed off into the circulation, and form embolisms, or become sufficiently disintegrated to form merely capillary embolisms, or even to a great extent be lost in the circulation. This mass has usually eroded to some extent the arterial wall beneath it, and leaves an ulcer on the artery (Usur). This commonly leads to aneurism, either simple or dissecting.

Atheroma, and especially when it is general and tends to calcification, may become general, and extend from the aorta down into the coronary vessels, up into the vessels within

the encephalon,—these, indeed, are commonly affected, and in fact over the whole arterial system to its termination in muscular arterioles. The veins, and especially the large veins, are liable to become atheromatous; but venous atheroma is never so marked as is the disease in the arteries.

Causes.—The exciting cause of atheroma is usually a cachexia, and it is found along with chronic Bright's disease, gout, syphilis, cancer, and chronic alcoholism. It is connected with such chronic affections as are regarded rather as blood diseases. The endarterium has no blood-vessels of its own, but derives its nutrition from the blood-current rushing through it (Rindfleisch). Thus, whenever the blood is altered in its properties, this alteration leads to tissue-growth, the lowest form, that of basement membrane, being most readily affected. It seems that the lower the form of tissue-life, the more prone it is to take on abnormal growth. Thus alcohol, gout, and syphilis poisons are tissue-irritants apparently. In syphilis we recognise a tendency to form masses of young connective-tissue in various tissues, muscle, subperiosteum, brain, &c., and the process of forming gummata in the heart may proceed up into the scanty muscular tissue of the aorta, lying beneath the intima. But unquestionably the great exciting cause of atheroma is *strain*. Thus we find it at points of most tension, at branches or bifurcations, at the coronary orifice; we find it in the pulmonary vessels behind mitral disease, and in the venæ cavæ after tricuspid regurgitation; as also in veins when an artery opens into them. We find it in the pulmonary valves following on the accentuation of the second sound brought out by strain. We find it in the aortic valves in hammer-men, and we find it, above all things, in chronic renal disease. The connection of atheroma with gout is well established. Rindfleisch, in section 215, states that gout and endarteriitis have a common ætiology, and are, therefore, commonly found together. How this connection exists we saw in the last chapter; how imperfectly depurated blood in renal disease led to vaso-motor spasm, hypertrophy of the muscular arterioles and the heart, and excessive distension of the elastic tubes connecting them. Strain is the great exciting cause of this parenchy-

matous inflammation called atheroma. Chronic disease of the lining membrane of the circulation is too intimately associated with strain to admit of doubt as to the existence of association, and it is impossible to refer their co-existence to mere coincidence.

Dilatation of the Aorta (Erweiterung).—This affection is allied pathologically to aneurism, but differs from even fusiform aneurism, in being rather a generalised than a localised affection. The aorta becoming generally atheromatous is thickened, its surface roughened, and its elasticity impaired. The first effect of atheroma is to diminish the calibre of the vessels by its own thickness, obviously enough. But as the arterial coat becomes altered dilatation takes place. The heart is usually hypertrophied, and the distending force of the blood-current considerable, the degenerate artery is distended forcibly, and its recoil is not quite perfect. Thus in time with the repeated distension of every systole the calibre of the vessel is altered, it becomes generally widened and dilated. This is especially the case with the upper portion of the aorta, and especially on the outer surface of the aortic curves.

Symptoms and Signs.—This affection is not of a demonstrative character, and is rather detected by the physician than a cause of suffering to the patient. There is a decided increase in percussion dulness across the aorta at the second right costo-sternal articulation: there is also a harsh systolic murmur, especially when free from aortic regurgitation; this is due to the roughened surface, and the blood-current becomes audible over it, as a murmur. There is commonly, too, a delay in the pulse of the extremities. But the most certain diagnostic sign is to insert the finger in the sternal notch, at the same time telling the patient to thrust his head forward. Then, the aortic heave becomes distinctly perceptible, and the murmur may be felt as a thrill. This is not likely to be confounded with an aortic aneurism.

This condition is usually accompanied by cardiac hypertrophy; but sooner or later the impaired aortic recoil leads to imperfect coronary circulation and to degeneration of the heart walls.

The affection is very commonly found along with aortic valvulitis, both resulting from the same cause. The affection is also usually found either in the subjects of chronic Bright's disease, or in " strikers," &c. It is one of the outcomes of arterial strain, and as such is rarely found alone.

Prognosis.—There is no very favourable prognosis here, and the most that can be hoped for is, a tardy downhill course. The tendency is to death from cardiac failure, due to tissue degeneration. The condition is rarely confined to the aortic arch, though most marked there, and in this generally atheromatous condition, cardiac failure may be anticipated by some intercurrent disease carrying off the patient.

Aneurism. Varieties of Aneurisms.—Aneurism is a more localised affection, and is due either to localised disease causing the wall to yield, the old true aneurism, or possessing all of the arterial coats; or it may result from an atheromatous patch undergoing fatty degeneration and disintegration, forming an ulcer, then it was called a false aneurism, *i.e.*, not possessing all the tissues. This aneurism may be simply bulging, or it may be dissecting, and burrow under the edges of the ulcer, betwixt the arterial tissues, and ultimately point and burst at some point at a comparative distance from the the ulcer in the inner coat. At other times the aneurism is more general, and is fusiform or spindle-shaped, or annular, a bulging all around the aorta; this last form of aneurism has been supposed by Rokitansky to take its origin in vaso-motor paralysis of a portion of the vessel. The seat of aneurism may be any part of the artery including the sinuses of Valsalva.

The pathology of aneurism is that of the atheromatous process with some effort, to rupture a weakened spot. This is not always the case, as in the dissecting aneurism from an atheromatous ulcer, and in the case of lost vaso-motor contractility. It has been always, up to a recent period, asserted that aneurism never occurred in an artery unaffected by pre-existing disease. The occurrence of purely traumatic aneurism from falls and accidents has of late caused some to adopt the view of the causation of aneurism by a shock or

blow over the vessel during its systole, or when fully distended by ventricular contraction. This view has been supported by Dr. Clifford Allbutt with his usual able advocacy, and he holds that when so distended, the brittle inner coat is easily cracked and an aneurism formed. When once formed, there is not much question as to its progress; every systole, especially under excitement, adds its mite; for the pressure of the blood being equal on all parts of the arterial system, the weakest point will always most readily yield. The pulsating sac may preserve its roundness, or become nodulated, or pouched, burrowing among the tissues when so pouched, and advancing by erosion when round. The course may sometimes be indicated by pressure on some organ; there may be difficulty in breathing, from pressure on the air tubes; or in swallowing from pressure on the œsophagus; or dropsy from pressure on the venæ cavæ, sometimes the dropsy being confined to one or other extremity from pressure on one vena cava only. Or a nerve may be compressed, and the recurrent laryngeal, which curves round the aortic arch, is especially apt to be compressed, producing hoarseness, aphonia, or the "ringing cough," said to be diagnostic of aneurism. Or some large arterial branch may be affected, and the pulsations of two corresponding arteries, as the radial for instance, be rendered unequal. Pressure on the ganglionic nerves is supposed to affect the size of the pupil of the eye, on the same side as the aneurism. Pain, ever constant in the spine, is suggestive of erosion of the spinal column by an aneurism, and Walshe lays great and deserved stress on this point: constant pain in the spine without apparent cause being ever significant.

Progress and Terminations.—The progress of a thoracic aneurism is untoward, and leads to death, variously. Rupture of the sac by a blow may anticipate erosion of the coats; dyspnœa may prove fatal; or the patient die of inanition from the difficulty in swallowing. Erosion of the spine may lead to spinal meningitis, and erosion of the sternum to external hæmorrhage. We know little or nothing of the result of pressure on the thoracic duct. General ill-health, dropsy, &c., may gradually wear out the patient. Rupture

may occur into various parts not leading to sudden death; when into either mediastinum it is apt to be quickly fatal. Even erosion of the bronchial wall and hæmoptysis has been followed by arrest of the hæmorrhage by a clot, the hæmorrhage not returning for some time, but proving fatal finally; this was the case with the famous Liston. The rupture will sometimes take place into the veins, arteries, or chambers of the heart itself, of which instances have been collected by Dr. Peacock; by far the largest proportion burst into the pulmonary artery, amounting to three-sevenths of the whole collected cases.

Diagnosis.—The diagnosis of an aneurism, in nearly every case, depends on the peculiar symptoms of each case, their grouping, and relation to each other. When deep seated we can get no aid from inspection, palpation, or percussion, unless it be deep seated, comparative dulness, a not very trustworthy sign. There is usually a distinct bellows murmur (Blasebalggerausch), which may at times obscure the normal sounds of the heart, and at other times leave them distinct; in this latter case the diagnosis of aneurism, or of tumour pressing on an artery, is cleared from any heart affection producing murmur. This is the only positive sign. Delayed pulse at the wrist is of some diagnostic aid. The symptoms derived from pressure will often throw much light on the scanty positive evidence of the existence of thoracic aneurism. The following direct expression by Niemeyer will illustrate the difficulty of diagnosing deep-seated aneurism:—"As long as the aneurism remains enclosed within the thorax, without touching its wall, diagnosis is not assisted by physical examination." When the aneurism approaches the thoracic wall then it may be seen pulsating in the inter-costal spaces, or felt by a thrill, the "fremissement cataire." Its diagnosis is no longer difficult when the thoracic parietes are reached. Dr. Peacock says, "The patients, who are the subjects of aneurism of the ascending portion of the aorta, are most commonly of the male sex, and at or about the middle or more advanced period of life. Not unfrequently they have been the subjects of rheumatism, and have been addicted to spirit-drinking and

habits of intemperance. Most usually they present the common cardiac symptoms—dyspnœa, palpitation, and tumultuous action of the heart, dropsical symptoms, and signs of engorgement of the lungs and parenchymatous viscera. These symptoms sometimes commence insidiously, and advance gradually; in other cases they occur somewhat suddenly, and as the result of some injury or strain." The diagnosis of thoracic aneurism, as long as deep seated in the thorax, is to be founded on the especial symptoms of each case, and not by any rules which can be laid down.

Prognosis.—So rarely does a thoracic aneurism end in recovery, that the prognosis is necessarily very unfavourable. Life may be maintained for some time by proper care, but it is rather with that object, viz., retarding the progress of the case, than in the hope of cure, that treatment is adopted. Still as success is possible, even though highly improbable, it should be borne in mind as a possibility.

Treatment.—The treatment of aneurisms must be conducted on the principle of avoiding what would add to the size and volume of the aneurism, or tend to rupture it. Thus great quiet of body and mind is absolutely indicated, avoidance of excitement, exertion, or effort must be insisted upon. No straining at stool must be permitted, and the bowels must be kept regularly open, and the patient must be warned against straining, and told, if the bowels do not act easily, to wait some time and make another attempt, anything in preference to straining. All causes which would hasten the pulse, as alcohol, &c., must be carefully avoided.

For the indications in the treatment of aneurism are not precisely the same as in the treatment of disease of the heart itself; there ventricular contraction must be maintained, here every increase, either in number or power, of the heart's contraction adds to the mischief already inaugurated. A few beats more or less each minute makes up a grave sum total in the day of the number of times this sac has been distended, and the pulse may be well kept as slow as well as low as possible. This has been attempted by the administration of digitalis until the symptoms of poisoning by it—small, feeble, slow, irregular pulse, from abnormal contracted

condition of the ventricle—are induced. Another plan is to lower the ventricular contractions by the administration of aconite; a preferable plan to the digitalis poisoning.

The old plan of treatment of Albertini and Valsalva by bleeding and starvation has not now many advocates. A plan of treatment has recently been adopted, especially by the Dublin School, with very encouraging success. It consists in a rigorous adhesion to the recumbent posture, to reduce to a minimum the effects of pulsation, the effect of the horizontal posture in reducing the number of pulse-beats per minute is well known: in addition to this purely physiological treatment, iodide of potassium is administered in full and large doses. The results of this treatment are sufficiently encouraging to make further trial of it desirable; for if the success be limited, the prognosis of the disease must be taken into consideration. Astringents administered internally, with the hope of coagulating the contents of the aneurismal sac, are not now much resorted to, and the same may be said about opium. In the severe pain often caused by an aneurism opium, the subcutaneous injection of morphine, or other narcotic, are very useful. Here the pain is due to pressure on a nerve, and to relieve it there are only two feasible plans, to remove the pressure, not very practicable here, or to deaden the susceptibility of the nerve centres, the only effective plan.

When the aneurism has reached the chest wall, other plans of treatment are feasible. To relieve pain and violent pulsation in the sac, the Germans are fond of ice, pounded, and applied in an india-rubber bag. To attain the same end lotions of lead and opium have been resorted to, not without effect. To attempt coagulation of the contents of the sac, galvano-puncture has been used. This method was first used by Petréquin, and has since been tried by many. It is rather adapted to hospitals than general practice, and the treatment is yet rather experimental than established. When the aneurism has fairly eroded its way through the chest wall, and formed a pulsating tumour on the surface of the chest, a cover of leather, moulded to the tumour, sheet-lead, or other material, may be applied to reduce to the risk of

blow or other accident. When so protruding these aneurisms have been known to be opened by the lancet, chiefly by irregular practitioners, and it is somewhat satisfactory to know that these ill directed surgical efforts were not immediately fatal. This was due to layers of fibrin under the sac wall; and when the sac and skin are ultimately opened by ulceration, sometimes life is prolonged by a clot plugging up the orifice, just as in Liston's case a clot in the bronchial orifice prolonged life for some weeks.

The no less interesting subject of aneurisms of the descending aorta, both thoracic and abdominal, cannot be taken up here, as not directly belonging to the subject under consideration.

The veins near the heart are rarely themselves affected, except by atheroma, which neither leads to rupture nor aneurism. Tumours, simple, malignant, or aneurismal often, however, press on one or both venæ cavæ. The dropsy, confined to the district of the great venous trunk pressed upon, will often indicate the nature of the cause of that localised dropsy. When general, however, it cannot be distinguished from dropsy arising from tricuspid failure.

CHAPTER XV.

Malformations of the Heart.

The heart is liable to various malformations, which often interfere seriously with the performance of its function. At one time the prominent symptom, cyanosis, was applied as a designation covering each malformation. More exact research has demonstrated that these malformations arise in four ways, viz. :—1. Imperfect evolution. 2. Peculiarities of the fœtal circulation remaining. 3. Diseases during fœtal life. 4. Mere anomalies.

1. *Imperfect Evolution.*—This may amount to total absence of the heart, a matter of little moment to physicians. Then the heart may remain of batrachian type, and consist of only one ventricular chamber, due to imperfection in the septum ventriculorum. The imperfection may vary from a small perforation to a complete loss of the septum. At other times the arterial stem remains uncleft, and the aorta and pulmonary artery are in one vessel. At other times, again, the stem is cleft, and the relative position of aorta and pulmonary artery transposed.

2. *Peculiarities of the Fœtal Circulation remaining.*—The foramen ovale may remain patent, and where there is also tricuspid stenosis the foramen ovale rarely closes. The Ductus Botalli may remain, and a communication betwixt the right ventricle and the descending aorta may exist permanently.

3. *Diseases during Fœtal Life.*—During fœtal life the heart is liable to endocarditis, and various malformations arise therefrom. As during fœtal life the circulation is mainly carried on by the right side of the heart, diseases then occurring are commonly found, if not always, in the right side. Indeed, primary disease of the right side of the heart is very

rare in extra-uterine existence; but when failure in the mitral valve has thrown the stress of the systemic circulation, to some extent, at least, upon the right side, it again becomes liable to disease. The most common results of fœtal endocarditis are stenosis of the ostia, pulmonic, tricuspid, or rarely aortic or mitral. This often takes place in the conus arteriosus, and not absolutely at the arterial orifices. In the auriculo-ventricular ostia the ring round the ostia is affected. Stenosis may affect the blood-vessels, and in the Pathological Museum of Vienna there are two cases, one of aortic stenosis below (immediately) the Ductus Botalli, the other stenosis of the lower vena cava, close to the heart. In both cases great vessels were found in connection with the internal mammary and the vessels of the dorsal muscles. It has been recently asserted that imperfection in the septum ventriculorum may arise from disease in fœtal life. Here it is supposed that a syphilitic gumma has existed in the septum, and the part, thus weakened, has given way and opened the ventricles into each other.

4. *Mere Anomalies.*—These may be found, as four semilunar valves for three, three flaps in the mitral, &c., but the most interesting is transposition of the viscera. Here the heart, along with the spleen, is on the right side, and the liver and ascending colon on the left. The position of the stomach is reversed, and the pylorus points left, and the cardiac orifice to the right. The lungs are transposed also. It is not recorded whether these persons were left-handed in life, or not. In the Vienna Museum there is an excellent specimen of this, and a second is being prepared.

These malformations may affect the heart's function most gravely, or not at all, according as to which is present, and to what extent in some of them. In some cases post-mortem examination alone detects them; in others they incapacitate the sufferer from an independent extra-uterine existence.

With neither of these, as physicians, can we have much to do, but in the numerous less extreme cases we may be consulted. The chief question is as to how far the children so afflicted are likely to live. Some short history of these unfortunates may not be out of place. As children, they are

blue with dark lips, and readily induced dyspnœa. They are usually of somewhat lowered temperature, little able to stand exposure to cold, and readily affected by changes of temperature. The periphery is liable to undergo changes, and the nose and lips are blue, while the fingers and toes are dark coloured and clubbed. The tips thicken, and the fingers especially are club-like. These sufferers may dwindle on to puberty which they can rarely accomplish, they seem too cold blooded to take on puberty with its accompanying passions; they are reptilian like, and the batrachian heart seems to involve a sort of batrachian existence. The mental manifestations are feeble, and the mind and body remain childish, even when the growth exceeds that of childhood. In a case which occurred to my friend, Dr. Elliot, of Carlisle (see Proceedings of the Royal Medico-Chirurgical Society, 1868), the young man had reached manhood, and was being educated for a missionary, when he died. The cyanotic signs first showed themselves when three months old, and yet he lived to the age of nineteen years and eight months. His heart was univentricular and batrachian, the septum ventriculorum being totally wanting. The subjects of congenital cyanosis are liable to suffer from venous congestion and its consequences, especially plethora abdominalis, and usually evidences of insterstitial growth of connective tissue are found freely in liver, spleen, and kidneys. Death may occur from the direct consequences of these visceral changes, or from some intercurrent disease, which the sufferers with weak hearts, and altered viscera therefrom, are incapable of successfully withstanding.

"Mere imperfection of the septum does not cause cyanosis, but is a harmless anomaly, which gives no evidence of its existence during life" (Niemeyer). And the possibility of such congenital imperfection must not be forgotten, when strange or anomalous heart affections present themselves.

Diagnosis.—The diagnosis of congenital malformation is usually not difficult, but the diagnosis of what imperfection you have to deal with is usually impossible.

The splendid collection of cases, with histories, in Dr.

Peacock's book must be appealed to for further information, not only as to diagnosis, but for other information on this subject generally.

Prognosis.—This is in accordance with the history given above, but observable congenital malformation, not being mere transposition, carries with it ever but a poor prognosis. A necessarily short life is surrounded with more than ordinary perils from intercurrent affections.

Treatment.—There can be no treatment more than which is merely palliative, and such measures may be used in each particular case as the necessities of the case may seem to indicate, or the good sense of the practitioner suggest. One thing is certain, these human reptiles must ever be warmly clad and protected against atmospheric variations of temperature.

CHAPTER XVI.

Concluding Chapter—Elements of Prognosis in Heart Disease.

General Elements of Prognosis—Embolism—Heart Disease in Thoracic Deformity—Heart Disease from Chronic Affections of the Respiratory Organs—Heart Disease and Phthisis in Young Persons—Valvular Disease and Phthisis—Clubbed Fingers—Reduplication of Heart Sounds—Persistency of Murmurs.—Conclusion.

The necessity for placing the remarks on prognosis in the concluding chapter is obvious, for all the different forms of disease must be considered, and the distinctive points touched upon, before any arrangement of the different factors of prognosis could be attempted.

Prognosis, of course, is the opinion of the medical man as to the probable course and duration of a disease, and can obviously exercise no control over the disease itself; unless that prognosis have been rashly and unwarily given to the patient in a manner to seriously shock or alarm him. There is no doubt that, in the case mentioned by Stokes, where, on hearing a cardiac murmur, a medical man announced to the patient, "Ah! I have just heard your death-knell," this unwarrantable speech had a very unfortunate effect upon the patient. Neither is it proper that the patient should be frankly told that "You will die suddenly some day!" as in a case well known to myself, and where the outspoken doctor has some years ago gone to his rest, and very suddenly too, while the patient lives on without any evidence of organic disease about him, hale and active, but his life simply poisoned by that rash opinion; and had he really had dis-

ease of the heart, as was supposed by the frank medical man, it is very probable the effect of this outspoken prognosis would have gone far to verify it.

Treatment, too, will often tend to verify a hopeless prognosis, possibly not warranted by the facts of the case. But putting aside these auxiliaries towards verifying a prognosis, there are certain signs and symptoms which will guide us in forming a prognosis. It will, perhaps, be best to give the indications for a gloomy prognosis, and, of course, the absence of them will always go far to warrant a hopeful prognosis; and to commence with the indications given by the heart itself.

Heart's Force.—The loss of impulsive energy in the heart, the impaired vigour of the ventricular contractions, is ever an unpleasant indication, and more decidedly so when the first sound is low or lost. The tendency to become broader, and the percussion dulness wider, is significant that the chambers are suffering from dilatation, and this is the more significant if previous hypertrophy has existed; for it suggests the probability that the hypertrophy is yielding, and a dilating process inaugurated, by degeneration of the muscular structure of the heart itself. This is often found to be the case where there is atheroma of the arteries and co-existing Bright's disease.

Rythm.—Taken in conjunction with loss of force the loss of rythm is suggestive; but loss of rythm alone may be a mere nervous disturbance. When, along with increasing loss of force, the heart's action becomes unrythmical, the condition is a grave one. Palpitation induced by slight effort is suggestive as indicating a tendency in the chambers to become distended or engorged. More serious is irregularity in the pulse on slight effort, whether combined with palpitation or not; but when with an irregular, or worse still, intermittent pulse syncope is induced by effort, then the prognosis is very bad. Intermittency, not nervous, but organic, is ever a serious indication, and indicates commonly structural disease of the heart-walls, while the existence of a number of rapid, small beats before the pause is usually indicative of dilatation.

Valve Failure.—Whenever a vavular lesion co-exists with evidences of muscular failure, and the presence of objective symptoms, the prognosis is worse, *cœteris paribus*, than when no such lesion exists. The implantation of a new cause of failure in the circulation on an old one, which has already taxed the recuperative powers of the system in establishing compensatory forces, is ever of grave significance prognostically. We know nothing of valve failure resulting from muscular failure only, and without disease in the valves themselves, as it is never so produced; Rokitansky saying that the valves stretch along with the muscular walls and the ostia, and Oppolzer and Kürschner stating that such valve failure is anatomically impossible; though Wilkinson King could produce tricuspid leakage in the heart when removed from the body. But of the opposite condition of muscular failure coming on after valvular failure has existed some time; we have unfortunately too many opportunities of seeing it. As to the prognosis to be arrived at from the seat of the valvular lesion, Dr. Peacock says (on the Prognosis in cases of Valvular Disease of the Heart) "The order in which these conditions should be placed, as indicating their relative danger, beginning with the more serious affections, would, therefore, be as follows:

Aortic Regurgitant Disease.
Mitral Regurgitant Disease.
Mitral Obstructive Disease.
Aortic Obstructive Disease.

The comparative rarity of serious affections of the right side of the heart, and their being usually combined with other defects in the conformation of the organ, make it difficult to estimate the relative danger which attends them, as compared with disease of the left valves." Dr. Peacock puts the positions correctly here, unless some doubt may be felt as to the comparative danger to life in the affections of the mitral. Combinations of valvular lesions, and the failure of one leading to disease of another behind it, especially must be regarded as ever of grave prognostic import, and the occurence of tricuspid insufficiency after mitral failure marks

an epoch on the case, and a date from which the downward process will be rapid.

General Power.—Impairment of general power, and the rapid oncome of dyspnœa, and the various objective symptoms of heart failure on slight exertion, are indications of bad omen. Often the patient will have spontaneously and instinctively, but unconsciously, assumed a very steady slow walk with great watchfulness against shocks, or any necessity for exertion; and there is a gait almost pathognomonic of heart disease, especially of structural decay. Along with this will commonly be found the evidences of mental impairment from cerebral anæmia. There is no aberration of intellect, it is simply amentia from the impaired supply of arterial blood. The patient's gait and walk are accompanied by imperfect power of thinking, and a want of self-confidence, and when these general indications are found along with the other indications of heart failure, the prognosis, as to life, is hopeless; and even as to time is very gloomy.

Loss of Arterial Tension.—This is ever of great moment in forming a prognosis, and the best external evidence of this loss, the diminution in the bulk of urine, is ever significant. This may be somewhat obscured by pre-existing kidney disease, but the falling off is still distinct enough, when failing power in the heart is lessening the pressure on the glomeruli of the malpighian bodies. The prognostic import of falling off in bulk in the urine is well known among the laity of the north of England, and is regarded as a symptom scarcely less significant than the appearance of dropsy. Thus popular opinion and the investigations of Traube coincide. The effect of increase in the bulk of urine after the administration of any agent, as digitalis for instance, is always hailed as a good sign. This is not a difficult subject to understand when we remember the clear connection betwixt arterial tension and the elimination of water on the one hand, and betwixt arterial tension and ventricular contraction on the other. Any improvement in the action of the organ, or pump, for its action is mechanical, which lifts the blood from the venous side over into the arteries, is at once felt in the increase of arterial tension.

Venous Congestion.—It is obvious that venous congestion and arterial tension are proportioned to each other inversely, increase in one lessens the other, when the capillaries transmit the blood more quickly to the veins, then the heart passes it again into the arteries. We get the veins too full and the arteries too empty. The effect of such agents as digitalis in contracting the capillaries, may aid in increasing arterial tension, by not letting the blood flow out so readily by them, as well as by increasing ventricular contraction. The loss of arterial tension, as shown by diminution of bulk of urine, is accompanied by venous congestion, as shown in the engorgement of the viscera, called plethora abdominalis; and when this has proceeded to the point of albuminuria, and the production of exudation tube-casts, the condition is serious indeed. Here the impaired circulation in the nutritive vessels of the kidney leads to imperfect elimination of urine salts, though the small bulk of albuminous urine may be of high sp. gr., and laden with urates; and this imperfect elimination leads to vaso-motor spasm, which further taxes the already enfeebled heart.* The presence of albuminuria and new exudation-casts is of the gravest import.

Dropsy.—Though dropsy may come and go again, its prognostic value is unmistakable; and when, with the symptoms of heart failure, œdema sets in over the arch of the foot, and round the ankle, and though beaten off for a time, returns again in greater force, each time more persistent, until, at last, firmly established, the indication is very serious. Here even the venous valves of the lower limbs are unequal to preventing venous engorgement, and water is effusing into the areolar tissue. The venous twigs are too full, and the watery constituents of the blood are escaping through the coats of the venules, indicating a debility in the centre of the circulation, of the gravest significance. When the venous dilatation has rendered the jugular valves incompetent, and the mere undulation or wave from the backward flow

* This is not a contradiction to the possible good of digitalis, whose action on the ventricle is also accompanied by action on the arterioles and capillaries. The peripheral circulation has been already dilated from the effect of distension of the heart acting on the vaso-inhibitory nerve.

on closing the tricuspid is converted into a pulsation, it is evident that the ventricular contraction is no longer entirely shut off from backward flow; and that there is a leak in the tricuspid, which is exposing the veins behind to the force of ventricular contraction, dilating them and rendering their valves incompetent, and thus increasing venous congestion; while the amount flowing back at every systole is so much less for the left ventricle to pass forward into the arteries.

These are the elements of prognosis considered generally, but each individual case will have certain peculiarities of its own, which the practitioner must take into his calculation. Thus necessity for exertion, for labour, makes the prognosis ever worse for the working population than the wealthier classes. The presence of any other old standing disease adds to the gravity. Something, too, depends on family history. In some families death is apt to be sudden, while in others the patient endures to the "bitter end." The mind and force of character affect the prognosis; and the mind, which can best maintain its balance, endows the owner with some degree of protection; while some minds never recover the shock of the most careful and guarded prognosis. Circumstances likely to excite the patient are very unfavourable; the excitement of war times, of important law suits, of exposure to annoyance or irritation are all bad. The great John Hunter felt his life to be in the hand of any one who should excite his choleric temper, and the result justified his prognosis. The liability to some intercurrent disease must not be overlooked; and an occupation involving exposure, and thus liability to cold or inflammatory affections, ever lessens the chance of life. In some cases there is great risk from rupture of one of the vessels within the encephalon, and the occurrence of cerebral hæmorrhage. While again there is another danger, which defies our calculations often, and though occurring without heart disease, is still more liable then to occur, viz., the formation of vegetations in the altered valves, or thrombosis in the ventricular chamber, and the occurrence of embolism.

The importance of diseases occurring from solid matters finding their way into the circulation, and ultimately lodging

somewhere and arresting the circulation, has only been recognised in recent years. These solid bodies form variously, but one end is common to all, viz., a floating in the blood-current until the calibre of the vessels no longer admits of further passage, and an embolism is formed. The embolon itself may arise, and commonly enough does, from the settling of fibrine on the surfaces of the valves; deposits on the valves, not vegetations springing from the valves, and these are very liable to form during endocarditis, when the two great factors, a fibrinous condition of blood and an altered condition of endocardium are found together. Clots may form among the columnæ carneæ and become dislodged. They locate themselves in different places according as accident in the blood-current determines. The straightest course is the most favourite one, and embolism is most commonly found in the left middle cerebral artery, causing the marked right side paralysis with aphasia so characteristic of an embolon. This sudden arrest of circulation fells the patient as with a stroke, and thus the cerebral affection differs in its onset from cerebral hæmorrhage. After passing the aortic arch, the clot may lodge in the branches of the cœliac axis, and lodge in the spleen, liver, or superior mesenteric. Small clots are very apt to pass into the renal artery and lodge in the kidney. Large clots may plug the iliac arteries or their branches. Small particles may cause mere capillary embolism. The consequences of an embolon are summed up by Cohnheim* as of four orders. The plug may remain quiet and without consequences; or gangrene and necrosis may follow; or infarction behind it; or formation of abscess.† To permit of the first there must be a possibility of collateral circulation being established.

The clot may lodge in the coronary arteries themselves. At other times the embolism may arise from the pulmonic circulation, and be the result of tissue-necrosis from a clot

* Untersuchungen über die Embolischen Processe. Berlin, 1872.

† Another consequence of embolism must not be overlooked, and that is certain material can, when lodged, excite a similar cell-growth to itself in the new locality; thus cancer-cells from scirrhus of the pylorus excite secondary cancerous growths in the liver, when they have found their way into the portal circulation and been arrested in the liver.

which took its origin in phlebitis. Embolism is very commonly found under other circumstances than those of heart disease, but is sufficiently associated with heart disease to be considered here. In guarding a prognosis, embolism, like cerebral hæmorrhage in hypertrophy, must not be lost sight of as a possibility.

Relation of Heart Disease to Phthisis in the Young.—In a few cases of caseous pneumonia in the young, the writer has noticed at the same time a dilated condition of the heart. In all these cases the prognosis was bad, and treatment was very ineffectual. The cases have not been sufficiently numerous to warrant any pathological inferences as to the connection of the two affections; but the opinion formed so far, inclines to regard the right ventricle as being more affected than the left; but the absence of a post-mortem examination, in the cases, leaves this subject very much in the dark. All that can be said on it with any certainty is the fact that the cases did very badly, and that in combined cases of phthisis and dilated heart the prognosis is very bad.

Heart Disease with Thoraic Deformity.—Rokitansky first pointed out how enlargement of the heart is commonly found in thoracic deformity arising from caries of the dorsal vertebræ. The altered relations of the thoracic viscera obstruct the flow of blood and evoke hypertrophy, sooner or later yielding, however, when the general health becomes infirm. In several cases cardiac dropsy in young people, with thoracic deformity, has been brought under the writer's notice, and in all the cases did badly.

In the several cases this breaking down occurred at a period when puberty would ordinarily be developed; but the cases were not numerous enough to furnish data sufficient for any positive expression. The dropsy was true cardiac dropsy, commencing from below upwards, and accompanied by objective symptoms of heart failure.

Heart Disease and Chronic Affections of the Respiratory Organs.—Affections of the respiratory organs frequently occasion diseases of the right side of the heart. Thus caseous pneumonia, we have seen, will lead to right-side

failure. The affections most markedly connected with right-side failure are emphysema, interstitial pneumonia (cirrhosis of the lung), chronic bronchitis, and pleuritic effusion. These affections are also concomitants of heart failure, and in many cases it is impossible to ascertain which came first in the order of time: prognostically it is not of much moment, as their concomitance is grave enough. But pathologically it is interesting to trace the connection, and the connection of the affections with heart failure, as sequelæ, is traced in Chapter IV; the opposite condition of their standing to heart disease in the relation of causes, deserves a few words. In many cases it is not very difficult to trace the order of the combination, while in others the affection of the respiratory organs is under care when the heart affection shows itself. The effect of emphysema in the compression of some vessels and in the dilatation and elongation of others, and thus obstructing the pulmonic circulation; of pleuritic effusion in general compression of the lung; of chronic bronchitis in arresting the respiratory changes, and thus leading to impaired blood flow, it is not difficult to comprehend.

Thus, in treating these affections it is always desirable to bear in mind the probability of right-side heart failure, and the occurrence of such failure warrants at once a grave prognosis. In such cases the course is apt to be rapid, and that it should be so is intelligible enough. The right ventricle is yielding and the obstruction remains as bad as ever, or even now aggravated by the failure in the right ventricle, and a smaller bulk of blood is passed over to the left ventricle; aortic distension and recoil are impaired, there is general impairment in the supply of arterial blood, and this, too, affects the heart itself. At the same time the distended right ventricle is acting more rapidly, because never emptied, the period of rest is much diminished, and the heart wall soon becomes degenerate and fails entirely. The tendency of affections of the respiratory organs to induce heart failure by early loss of compensatory growth, is thus intelligible enough. In other cases the right ventricle hypertrophies more perfectly and efficiently, and life is longer maintained; and in these cases the ultimately fatal morbid process is

somewhat different. The strain on the tricuspid valves induces valvulitis, the valve then becomes incompetent, and we have the consequences of tricuspid failure to deal with; and our aid under such circumstances is not of much avail. In chronic affections of the respiratory organs the certainty of right side failure in time, unless the patient be carried off ere it is induced, must ever render the prognosis very gloomy, and a prolongation of life by care; the avoidance of fresh attacks of disease in the enfeebled respiratory organs, and proper therapeutic aid, is all that can, at the best, be hoped for. The evidence of right-side failure having commenced no longer warrants anything but the most unfavourable prognosis, even as to time.

Chronic Valvular Disease and Phthisis.—While disease of the heart not only predisposes to lung disease of various forms, but is really followed by certain diseases of the respiratory organs as results, as consequences of itself, a somewhat curious effect has been pointed out by Rokitansky. He, as a pathologist, has noticed the fact that chronic endocarditis, or valvular disease, is rarely or never accompanied by any evidence of tubercle, or caseous pneumonia; that is, of any recent disease, for traces of such disease having existed at a time previous to the endocarditis are, of course, not obliterated by it. This fact of the absence of phthisis in chronic valvular disease had not entirely escaped Laennec himself. The curious association of chronic valvular disease with freedom from phthisis has been supposed by Traube to be due to the free transudation of blood-serum into the parenchyma of the lung in such valvular failure; a condition opposed to the condition favourable to caseous change in new cell elements which are due to a general deficiency of water in them.

Traube's hypothesis may not be quite satisfactory, but the fact, as indicated by Rokitansky, remains the same. On the other hand Lebert has regarded congenital pulmonary stenosis as a predisposing causes of phthisis. In five such cases which came under Dr. Peacock's notice, two had simple stenosis, and both died of phthisis; the other three had other defects in the conformation of the heart, and did not have

phthisis, and the lungs were much congested, but not tuberculous, at the *post mortem* examination.

Clubbed Fingers.—The peculiar thickening around the nails of the fingers, and, indeed, in the ends of the fingers generally, which has been denominated "clubbed fingers," was once thought to be pathognomic of congenital malformation of the heart, or cyanosis. Pollock has regarded it as sufficiently associated with chronic phthisis as to be to some extent distinctive. They are found, however, very commonly in cases of chronic valvular disease, and the thickening seems to depend on a proliferation of connective tissue elements in the part, due to blood-stasis, as seen in plethora abdominalis; and also to fulness of the free vascular distribution of the finger ends. The same fulness exists in the lips of young persons who have chronic valvular disease. The sign indicates most probably venous stagnation, and is thus found in chronic phthisis only as the result of obstructed pulmonic circulation and right side heart failure, with its sequel venous congestion. Its indication is essentially chronicity, and so far it throws some light on the past; but its use in aiding to calculate the future is not yet ascertained.

Though not quite in place, still it is equally difficult to see where they could be more appropriately introduced, I may here allude to two subjects not unconnected with prognosis, but still rather associated with the diagnosis of heart failure, viz., (1) reduplication of heart sounds, and (2) the temporary cessation of murmurs. Both are subjects which cannot be overlooked, and yet furnish us with no real information of practical utility.

1. Reduplication of heart sounds is occasionally heard very distinctly, and, though rare, is equally unmistakable when occurring. This phenomenon has had a great deal of attention paid to it, but its value is yet undetermined. Bouilland called it "une signe de luxe," sufficiently expressive of his opinion and experience. Nor has the most recent systematic writer on heart disease expressed himself any more favourably to its indicative value. "In its pathological import and diagnostic significance it is a sign of very little value" (Flint, p. 327). From the circumstances with which

it is associated, it would appear sometimes to indicate a lagging behind of the contraction of one side of the heart, from some obstruction offered to the flow of blood from it. But probably Flint's description is not far from being exactly true.

2. *Temporary Cessation of Murmurs.*—While murmurs are at times merely temporary in their character, as when due to anæmia, or spasmodic and irregular action of the musculi papillares; it is equally certain that valvular disease may exist without any evidence to auscultation, and that the murmur arising from it may be at times intermittent. We are not yet sufficiently acquainted with the temporary cessation of an organic murmur to say much as to its cause. Murmurs most commonly intermit at the auriculo-ventricular orifices, but even an aortic regurgitant murmur, the least likely of all, will occasionally intermit. When such temporary cessation occurs it is diagnostically troublesome, and may lead to a difference of opinion betwixt two medical men; not due in any way to lack of competence, or even to an active imagination, but simply to the fact that the murmur was audible at the time when the patient was seen by one, and inaudible when seen by the other. The chief indication, indeed, of this temporary cessation of murmurs is its value in leading us to be charitable to the mental processes of others. A murmur present when the patient consults a London physician and inaudible when listened for by a rural practitioner, might go far to inflict a severe blow to his reputation; and more so if again audible in the metropolis. Even under the best and most favourable circumstances, in such a case, the decision would probably rest on a calculation of probabilities, as to the flitting nature of murmurs versus the imperfect auscultatory powers of rural practitioners, and though the balance would descend in favour of the latter, the first must not be forgotten as a possibility. Another fact must not be overlooked, and that is the absence of murmurs in left side valvular disease after the tricuspid valve has become affected. In two well-marked cases which came under the writer's notice, most extensive aortic and mitral stenosis combined were found on *post mortem* examination,

along with tricuspid insufficiency from valvulitis. In the one a tricuspid murmur was audible over a very limited area at the ensiform cartilage, which passed away and was inaudible for some weeks previous to the patient's decease, and the left side double stenosis had never produced any audible evidence of its existence; in the other a faint diastolic murmur was heard over the third left costo-sternal articulation (the pulmonary area), but only at one spot, a most limited space in fact, and from this, and the marked evidence of right side enlargement, the diagnosis of probable pulmonary regurgitation was ventured in the face of its rarity. The *post mortem* examination revealed most extensive mitral and aortic stenosis, also allowing of regurgitation and great right side enlargement, hypertrophy with dilatation, and tricuspid valvulitis. From this it was evident that the murmur was aortic, and not pulmonary.

The general elements of prognosis must consist of a certain number of signs and symptoms variously combined, and rarely all existing together. Too much value must not be attached to any physical sign, trustworthy as they usually are, but a large and widely comprehensive view of every case must be taken. The effect of obstructed circulation is felt all over the organism, and the impeded working of each and every part must be taken into consideration. Death may threaten from heart failure directly in one case, from some distant consequential complication in another. The heart disease may be primary, and all else be but results of it, while in other cases it is itself secondary. The tendency, said to be on the increase, of a "specialist" to see everything as taking its origin in the organ to whose diseases he has specially given his attention, is not always a subject for a sneer; for if, of two men of equal attainments, the one turns his attention more exclusively to one organ's maladies and their sequelæ, while the other's attention is equally spread over the diseases of the body generally; it is no more than probable that the "specialist" can see some things not always readily apparent to the other; if it were not so, the argument would equally apply against a separate profession of medical men. But in the consideration of the various

consequences which follow any failure in the circulation, even to the periphery of the system, the recognition of heart failure and its importance is very necessary. The deprivation of arterial blood and the presence of venous blood in excess affects every organ and its functional working. The dependence of other affections, apparently primary, or some unrecognised failure of the heart, is more common than our self-pride as to diagnostic powers would sometimes admit. Chronic trouble, laid to the charge of a liver whose working is not quite "what it might be," will be found often to be due to a failure in the heart hitherto unrecognised. Uterine troubles and renal disease are alike, at times, the result of impaired circulation. Cerebral disorder, with corresponding psychical manifestations, is now known to be a result of defective supply of arterial blood and of venous congestion. While it is certainly undesirable to attribute more to the failure of power in the heart than can be fairly so attributed, or, in other words, to survey pathological anatomy from the stand-point of the circulatory centre; a performance containing within itself probably the same elements of failure as the survey of the universe, in astronomy, from the earth as a stand-point did; and so compelled all calculation to be made from an ideal centre for observation —a process which might be advantageously copied in practical medicine—it is equally certain that the importance of the recognition of heart disease and its consequences, and of the means of relieving it, is not generally fully appreciated.

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